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BULLETIN

OF

THE NEW YORK ACADEMY OF MEDICINE

MARCH, 1931

INCORPORATED 1851

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ANNUAL GRADUATE FORTNIGHT

APPENDICITIS

EUGENE H. POOL

Attending Surgeon, New York Hospital

It is beyond question, I think, that the appendix has caused more suffering, sickness and death than any other single structure of the human body. Yet it is so small and apparently so insignificant that its importance was not recognized until relatively recent years.

When the appendix is acutely infected its peculiar blood supply favors necrotic areas through its thin wall. Its floating and unprotected position in the peritoneal cavity favors spread of infection through that great area. Its generous circulation leads to early and intense reaction of the system to absorbed toxic products. Its structure embodies two tissues which elsewhere are frequently subject to chronic inflammation, namely, mucous membrane and lymphoid tissue; yet careful analysis, both gross and microscopic reveals nothing in either of these that might be interpreted as the source of a persistent chronic infection, such as is noted in connection with the teeth, tonsils, seminal vesicles or gall bladder. Whereas the appendix is a part of the intestinal tract, it does not participate in the digestive processes; in fact, in man it has no functional activity.

Our motive is a clinical not an academic consideration of

*Delivered October 23, 1930.

appendicitis. We are called to the bedside of a prostrated patient or asked to pass on a patient who walks into our office. In accordance with this objective, only three broad subdivisions need be discussed—chronic, acute and recurrent.

The usual conception of appendicitis is that inflammation of the organ is due to one of two causes, first, interference with the blood supply which may occur from pressure of a foreign body, as a fecal concretion, or distention from blockage of the lumen; second, a lesion of the mucous membrane which allows microörganisms to penetrate and proliferate. It is pictured by one author that if such factors are severe and sudden the acute type results—if they are mild but continuous or recurrent the chronic form occurs. This conception of the acute may be accepted. It is likewise true of the recurrent, which consists in intermittent attacks of variable intensity. It is immaterial how slight the clinical manifestations may be, there is nevertheless an acute reaction or inflammation as the underlying basis of each attack. Although recurrent appendicitis is usually classed as chronic, it is more properly a subdivision of acute.

The term appendicitis indicates inflammation. But we rarely, if ever, see an appendix which suggests an active chronic inflammatory process. At times, the organ is adherent to or incorporated in the wall of the cæcum. At times it shows sclerosis, scars, or a lumen partially obliterated, even with a definite stricture. Such conditions indicate an antecedent inflammatory process. But usually the organ is freely movable, not congested until traumatized and only shows a bend or kink which seems to excuse its removal. In other words, most of the chronic appendices show no gross evidence of active inflammation, though a certain proportion present signs of an inflammatory reaction at some previous time.

Symmers has made a careful microscopic study of several thousand appendices removed at operation. He reports definite changes in about 10 per cent, which is a very low percentage. We will summarize his findings:

The serosa pale and smooth; on section lumen diminished in size or occluded, mucous membrane thickened and swollen (pale), and in it pin point cream colored bodies which proved on microscopic examination to represent lymphoid follicles. In some, especially older patients, the appendix reduced to a pale cordlike structure, with a few atrophic lymphoid follicles and almost complete connective tissue replacement of the mucosa and obliteration of the lumen.

He assumes that the lymphoid changes are part of a general lymphoid peculiarity of a status type and the sclerotic secondary to this. Whereas this lesion which represents lymphoid hyperplasia is usually classed as chronic inflammatory, it is reasonable to assume that it is simply a local manifestation of a general peculiarity of the lymphoid tissues which occurs in some individuals. Under such an assumption this group is eliminated from chronic appendicitis.

We have shown that chronic appendicitis, strictly speaking, rarely, if ever, occurs—yet the public and even some of the profession have a loose and distorted idea of the term and of the indications for and results of removal of the organ.

We have tried to analyze the reasons which lead to the removal of so many "chronic appendices." They may be classified as follows:

Prophylaxis, i.e., to eliminate the danger of an acute attack.

Phobia, i.e., fear of an acute attack.

Dysfunction.

Discomfort.

Let us examine and criticize these:

On the basis of a few indefinite symptoms, as discomfort and vague digestive disturbances, the clinician is prone to visualize an appendix which is chronically inflamed. The patient is said to be suffering from chronic appendicitis and appendectomy is performed. Pain or discomfort, the most frequent of the symptoms, are often caused by gas in

the cæcum, resulting perhaps from a spastic colon; the patient is usually suffering from a phobia or fear of appendicitis. The appendix often is not diseased and the real value of the operation is the prevention of an acute attack and laying at rest the fear of such a development. "Chronic appendicitis" has become a friendly term by which functional derangements of the intestinal tract may be explained. Whereas, such symptoms as chronic constipation, colicky pains along the course of the large bowel, intermittent attacks of diarrhœa are usually purely functional derangements, they excite efforts to explain them by some definite lesion of an inflammatory, neoplastic or mechanical nature which can be corrected by surgical measures. In so far as this attitude leads to thorough study of the case, it cannot be too highly commended. But, unfortunately, chronic constipation and pains in the right iliac fossa often lead to unnecessary operations, such as removal of a so-called chronic appendix, repair of an incompetent iliocæcal valve, plication of a redundant or distended cæcum, freeing of kinks or bands, side tracking, as ileosigmoidostomy, and even resection of the colon. The symptoms usually persist after such procedures which are in general ill advised efforts to correct chronic constipation and its associated disturbances by the short cut of surgery.

Indications—When there is strong evidence of a single recent acute or subacute attack, or repeated suspicious attacks, it is wise to remove the organ. It is fair to assume that an acute attack threatens. Its prevention is definitely indicated. This is especially important when the habits or work of the patient separates him from surgical contacts. But such cases must be classed as recurrent appendicitis, not as chronic. It must be emphasized that operation should not be delayed when there is definite evidence of an acute inflammation even if this is apparently slight.

The appendix is a menace, especially in the young; with advancing years statistics show that the incidence of appendicitis drops rapidly and progressively. In the young, one is justified in taking a somewhat radical attitude.

It is difficult, indeed impossible, to understand how such an organ, in the absence of a real lesion, can exert an influence upon the general health of the patient, upon remote organs as the pylorus or upon the functional activity of the intestine.

On the other hand, as Alvarez and others have shown, irritation of the cæcum as the result of disease of the appendix, which may be due to inflammation or mechanical causes as stone or stricture, may produce all grades of back pressure from a slight iliac stasis to the vomiting of large amounts of intestinal fluid. The degree depends upon the intensity of the irritation. In the interval between attacks there are no symptoms; in a subacute attack there may be loss of appetite, nausea and some gastric or duodenal stasis and in an acute attack there may be persistent vomiting. It is thus evident that recurrent acute or subacute attacks of gastro-intestinal disturbances may often be cured by removal of the appendix. Yet considerable caution must be exercised when the temptation arises to remove a so-called chronic appendix to cure chronic gastro-intestinal disturbances. The appendix should be deemed responsible only after careful exclusion of other possible factors; and then it should be pictured not as a chronic inflammation but as some mechanical disturbance and probably dependent upon previous inflammation.

Pain in the right iliac fossa may be due to many factors, only one being the appendix. Either inflammation or obstruction of its lumen may be the cause. Persistent pain or discomfort in this region is probably not due to a chronically inflamed appendix, since such a lesion rarely, if ever, exists. One may properly visualize a localized discomfort as the result of increased peristaltic action (colic) due perhaps to inspissated fecal contents. Occasionally operation reveals such a condition and the patient is relieved by appendectomy. But this is unusual and removal of the appendix is likely to be disappointing in relieving persistent localized discomfort or pain.

Symmers concludes that there is a clinically recognizable syndrome referable to lymphoid hyperplasia of the appendix, marked by repeated attacks of appendical colic, occurring at intervals of days, weeks, or months, and is encountered most frequently in children or young adults, probably peculiar to subjects of status lymphaticus. In such cases the clinical indications for operation are not imperative, and the histology of the appendix shows that the changes do not tend in the direction of perforation but towards fibrosis.

In the course of laparotomy, the appendix should ordinarily be removed if there are no contraindications. This step adds little in time or risk. It removes a useless and potentially dangerous organ and often avoids a second operation, for the reason that not infrequently, especially after pelvic operations, adhesions cause pain in the right lower abdomen. If the appendix has not been removed there is doubt as to whether it is the cause, and in consequence, the patient may be subjected to another operation.

Our discussion of chronic appendicitis is not a quibble as to phraseology. It is, therefore, well to recognize that the lesion, Chronic Appendicitis, strictly speaking, does not exist. Symptoms are due either to recurrent acute attacks or to mechanical disturbances usually resulting from acute attacks. By reason of failure to grasp this many unnecessary operations are performed; too much in the way of symptoms is attributed to the organ; and too much is expected from its removal.

Diagnosis. Before even the relatively innocuous operation of appendectomy is undertaken a careful study should be made lest some lesion—the real cause of the symptoms—be overlooked and discovered later. This is not of infrequent record. Moreover, it must be remembered that the cæcum and appendix occasionally are not situated in the right iliac fossa. The cause of such an abnormal situation may be complete transposition of viscera, non-rotation, non-descent of the cæcum or long meso-cæcum. The anomaly must be borne in mind. It is wise, therefore, in chronic cases, especially in children, to determine the position of the cæcum and appendix by roentgenogram before operation is undertaken. While infrequent, it is an em-

barrassing operative experience to find the cæcum and appendix in the left iliac fossa after a prolonged and agitated search for them in the usual situation.

X-ray studies give little, if any, information as to whether an appendix should be removed. Such questions as failure to accept barium, retention of barium, segmentation, fixation, etc., are often emphasized but are of no real value.

Assuming that this paper will reach the practitioner rather than the surgeon we will omit technical details such as type and position of incision, extent of exploration and post-operative complications.

The important feature is differential diagnosis. Although this cannot be exhaustively covered, we can touch upon some of the most significant features and some which are not generally appreciated.

Frequently an appendix is removed when the trouble is entirely dependent upon a pelvic lesion, ureteral stone or stricture, disease of the gall bladder or duodenal ulcer. Proper consideration and study of the case should avoid such an error. One feature* is of diagnostic value in ureteral colic; ordinarily there is cremaster spasm which is usually associated with the other phenomena of the so-called "subinguinal syndrome" as hyperæsthesia limited to the ureteral skin triangle of the thigh.

Occasionally tuberculous peritonitis, spinal cord lesions, and neuroma of the 12th nerve are mistaken for chronic appendicitis. No rules can be laid down for the diagnosis of these.

Very frequently operation is performed when no lesion exists; this is peculiarly true in young Jewesses. We have found in the follow-up records of the Second Surgical Division of the New York Hospital for ten years (1916-1926) that the cases operated upon for chronic and recurrent appendicitis (unfortunately no differentiation was

*Supplement: *A. J. S.*, n.s. VIII, No. 2, p. 490.

made between these) showed a symptomatic failure is approximately 12 per cent. In analyzing these failures 125 cases were studied. The poor results were largely in young Jewesses; girls who were working in sweatshops, whose homes were poor, whose food was made up largely of sweets and sodas, whose exercise was practically nil, whose bowel habits were irregular, and whose occupation predisposed to pain on the right side, since many of these girls were operators and used the right foot in working the machines. These patients complained of indefinite pain in the right lower quadrant. After appendectomy they were free from pain for a short time and then it recurred. In the study of the post-operative failures, careful histories and exhaustive physical examinations were made. It was found that by diet, regulation of the bowels, creation of better habits, and improved hygienic conditions, much could be accomplished. Accordingly this type of patient with all the symptoms and signs suggestive of a chronic appendicitis, is now referred to the medical clinic and social service department, and operations have been much less frequent.

One of the most serious mistakes is in overlooking a real lesion of the cæcum, as tuberculosis, actinomycosis or carcinoma. Localized tuberculosis of the colon is of more frequent incidence than is usually supposed. Far too often it goes unrecognized. The most frequent sites are the cæcum and ascending colon. While several types of lesions occur, the most significant for the surgeon is the hyperplastic. This is a slow chronic development with marked thickening of the intestinal wall and relatively little ulceration. Symptoms develop when the lumen or the peristalsis of the bowel are affected. They are abdominal discomfort or colic in right lower quadrant, tenderness, diarrhœa or constipation. Constitutional symptoms and the presence of tuberculous foci elsewhere are suggestive.

Tuberculous mesenteric lymph nodes in children cause pain, tenderness and spasm which suggest appendicitis. X-ray often helps the diagnosis in showing calcium deposits.

Actinomycosis, a microörganism of the fungus variety, occasionally penetrates from the intestinal tract. The ileocæcal region is the usual site. A slow progressive inflammatory reaction causes marked thickening of the intestinal wall. In the early stages the lesion may readily be mistaken for carcinoma, hyperplastic tuberculosis, or chronic inflammation dependent upon the appendix.

The colon is a frequent site of cancer. The neoplasm ordinarily is limited in extent, slow in growth and late in metastasizing. Cases are often not recognized until a mass is palpated or signs of intestinal obstruction are present. Yet there are usually suggestive clinical features which should lead to investigation, correct diagnosis and relatively early surgery. These are blood in the stools, abdominal discomfort and derangement of the bowels.

The diagnosis of carcinoma of the colon as well as the two lesions previously cited can be made readily by roentgenogram following a barium meal or enema.

It is important that the possibility of cancer be considered in all cases presenting indefinite abdominal symptoms so that early studies may be made. The trouble is that we are too prone to jump at some other diagnosis and not weigh all the possibilities.

We have had three cases—one in the cæcum, one in the ascending and one in the transverse colon—in which cancer was not suspected before operation, the diagnosis being respectively, duodenal ulcer, cholelithiasis and chronic appendicitis. In all of these roentgenogram of the lower bowel should have resulted in a correct diagnosis.

To summarize:

Chronic appendicitis, as generally employed, is a misnomer. It rarely if ever occurs. The appendix is not the source of a chronic focal infection, it rarely is responsible for chronic symptoms referable to the gastro-intestinal tract. When there is evidence of a single recent acute or subacute attack or repeated suspicious attacks the organ

should be removed. Under other conditions one should be extremely cautious as to diagnosis and in general follow conservative measures before considering appendectomy for disturbances of the gastro-intestinal tract or discomfort in the right iliac fossa.

The pathologist is at a loss to describe the innumerable non-acute appendices which are referred to his laboratory. He cannot report them normal without causing ill feeling; he is practically forced to report "chronic inflammation." Yet there is no such lesion.

It is suggested that the pathological reports be somewhat as follows:

1. Evidence of recent inflammation.

2. Evidence of previous inflammation (scarring, stricture, obliteration, etc.).

3. Evidence of lymphoid hyperplasia.

After this somewhat negative presentation of chronic appendicitis we approach the acute with firmness and conviction.

Here is an acutely sick patient; here is a definite lesion; here is serious danger to life if the diagnosis is not promptly made with timely surgical intervention.

The only question then is whether the patient has appendicitis. If so, immediate operation is imperative. We will not even refer to the Ochsner treatment of delay which is of very limited applicability. It is likewise unnecessary to enter into the diagnostic details of a typical case. But some cases are enveloped in uncertainty and these are worthy of consideration. Our enumeration of the possibilities of diagnostic errors must not be attributed to erudition but to woeful accumulation of bitter experience. Nice judgment is often necessary and this, rather than technical ability constitutes the difference between the real surgeon and the artisan or mechanic.

Probably the most frequent and serious mistakes occur in children in whom the lesion is often far more advanced than the symptoms and signs suggest. Moreover, in infants and children early pneumonia is often diagnosed as acute appendicitis. In this connection we will quote from an article by Adams and Berger (*J. A. M.* A. v. 79, 1922).

"Diagnosis is often very difficult as is evidenced by the fact that of 145 cases of pneumonia over 17% were sent to the hospital as acute appendicitis. As histories are obtained usually with considerable difficulty, reliance must be placed mainly on physical signs and laboratory tests. Previous cold, cough or pain in chest are suggestive. Abdominal pain is difficult to evaluate. In pneumonia abdominal pain is in upper abdomen as a rule. However, there are two main differences: (1) In pneumonia, pain is more severe and persists. (2) In appendix, it is less severe and intermittent. Vomiting is very common in pneumonia. Therefore, pain and vomiting are of little help in differentiation. Headache, irritability and delirium are never present in appendicitis. More reliance is to be placed on the appearance than on anything else. Pneumonia cases are very sick, are irritable and fussy. Respirations are short, rapid, shallow and usually accompanied by expiratory grunt. The face is flushed. In pneumonia the temperature is usually above 102, in appendicitis usually below 102. There may be no lung signs. A slight change in percussion note (especially high in axilla) or in quality or in density of breath sounds are early signs of great significance. In this series 39% had no lung signs when admitted. All pneumonia cases had abdominal tenderness. But in pneumonia, tenderness is more superficial, more a hyperæsthesia and is usually in upper abdomen. In appendicitis, tenderness is deeper and more localized. Rectal examination with tenderness is strongly suggestive of appendicitis. Leucocytosis of 20,000, pneumonia more likely; below 20,000 appendicitis more likely."

Influenza often presents at its onset exclusively abdominal signs and symptoms—termed intestinal grippe—and

not infrequently this leads to the diagnosis of appendicitis, especially in children. Anæsthesia and operation under such conditions greatly add to the danger and discomfort, especially if pneumonia develops. But the pain and tenderness are not sharply localized and this together with leukopenia should cause suspicion of the true condition and excite caution.

Another confusing lesion in children is a type of erythema with visceral manifestations consisting in abdominal pain and tenderness with fever, but hæmaturia or eruption or both should suggest the true condition.

Just as in the erythemas so in the purpuras (Henoch's purpura) visceral lesions and manifestations may occur. Pain, tenderness, vomiting, etc., often lead to the diagnosis of appendicitis. Careful study will usually elicit some feature to lead to a correct diagnosis, for instance preceding joint pains for which antirheumatic drugs have been taken, and frequently purpuric spots may be found on the limbs.

Salpingitis is often difficult to differentiate and yet this is important since conservative treatment is here indicated.

Acute pancreatitis is sudden with marked prostration and a peculiar cyanosis.

Perforated ulcer usually gives some suggestive history, moreover the sudden onset as if the patient had been shot, with rigid scaphoid abdomen make a picture which can scarcely be mistaken in the early phase.

Undulant fever, due to the B. of Bang, has in some instances been mistaken for appendicitis and operation performed. Simpson and Fraizer (*J. A. M. A.* December 21, 1929, p. 1958) report three cases. Abdominal pain occurred in 11 of their 63 cases noted in or around Dayton, Ohio. Epigastrium in 4; right upper quadrant 4. In 3 cases sudden pain in right lower quadrant with fever, appendectomy was performed. I have seen a similar case. In all of these the correct diagnosis was made after operation by

agglutination test or blood cultures. In the case which I had the privilege of seeing, a young man of 21 complained of abdominal pain for about five months, located in lower right quadrant and for two months tenderness in lower right quadrant. Nausea after meals. Small frequent stools, chills for 3 months, loss of 12 pounds; 3 years previously he had chills, fever and prostration and was treated for malaria. The history should, in this case, have suggested the true diagnosis and appropriate tests should have been made before operation.

Diverticulitis of the colon has in recent years come to occupy an important place in abdominal surgery. The significant features are:

(1) Diverticulitis is a disease of adult life.

(2) The symptoms are similar to those of acute or subacute appendicitis, but the lesion being usually in the sigmoid, the symptoms are referred to the lower left quadrant, and are therefore like a left-sided appendicitis.

(3) The inflammatory process almost invariably becomes walled off and leads to abscess and not to diffuse peritonitis.

Among other lesions which may excite uncertainty are acute cholecystitis, ulcerative colitis, pyelitis, intussusception, ectopic gestation, pneumococcus and tuberculous peritonitis.

This summary gives but an imperfect impression of the innumerable lesions which may be mistaken for acute appendicitis or vice versa; as a matter of fact almost every acute lesion within the abdomen has at some time been responsible for such confusion.

We have thus reviewed in a very sketchy fashion some of the pitfalls which the clinician may encounter. The surgeon is faced with the final responsibility. In the event of an uncertain diagnosis he must decide whether the case demands immediate exploration or warrants further observation to arrive at an accurate diagnosis. He must plan

the exposure on the one hand for the case with definite diagnosis; on the other for the case which may demand more or less extensive exploration. He must weigh the contra-indications to operation; select the appropriate anæsthetic and then proceed expeditiously and coolly. The methods of handling an acute case are not standardized, yet statistics from large clinics are very similar. Our figures from the Second Surgical Division of the New York Hospital may be of interest:

From 1915 to 1930—1854 cases of acute appendicitis with 94 deaths, a mortality of 5.06 per cent. Among the causes of death were: peritonitis 51, embolism 2, pneumonia 2, pylephlebitis 1, sepsis 4. Over half of these deaths were due to peritonitis. It is fair to assume that almost all of these patients could have been saved by more timely diagnosis and operation.

Two series of cases were studied and recorded by my associates, Bancroft and Garlock:

1915-1921—584 Cases	1921-1928—755 cases
15 fecal fistulæ	12 fecal fistulæ
295 drained cases with	(3 required closure operatively)
45 herniæ—15%	264 drained cases with
205 sutured without drains	18 herniæ—5.4%
4 herniæ—1.9%	(no sutures in fascia or skin)
	236 sutured without drains—no herniæ

We cannot here enter into operative details. But it is well to emphasize that in recent years where drains have been employed in acute appendicitis cases, sutures in general have not been introduced except in the peritoneum—such open wounds granulate rapidly and as is shown by the statistics, post-operative herniæ are much fewer by reason of the fact that sloughing of the fascia does not occur.

BACTERIEMIA*

WALTON MARTIN

Attending Surgeon, St. Luke's Hospital

At the outset of a discussion on disseminated infection in which bacteria have been found in the blood stream, it is well to suggest limitations and to agree upon definitions; for the study of the spread of bacteria may be profitably discussed from many different points of view, and many of the terms used are applied with unlike meanings.

We shall not refer to the technique of the examination of the blood for bacteria.

The review of the clinical pictures presented in the dissemination of the different species of microörganisms will not be considered, nor the transient bacteremia such as occurs after operation, or manipulation in the male genito-urinary tract.

Nor will we discuss the phase in which microörganisms are found in the blood stream in the well recognized diseases, such as typhoid fever, pneumonia, relapsing fever, bacterial endocarditis, etc., except to use some of the information obtained to throw light on the sequence of events in various forms of septicemia.

It is the bacteremia occurring in the course of so-called septicemia, which, I believe I am expected to discuss. And I have taken for granted, that as practitioners of medicine and surgery, you have in mind renewing your acquaintance with some of the well known experiments which throw light on the sequence of happenings in disseminated infections. I assume that you will wish to hear the prognosis and the methods of treatment discussed, when positive blood cultures are found in patients lying gravely ill with irregular temperatures, with or without chills, rapid pulse, with or

*Delivered October 23, 1930.

without metastatic foci. For to quote Sacquepee, "the idea of septicemia corresponds both to a clinical syndrome of general disease and a biological fact constituted by the presence in the blood of one of the recognized pathogenic germs."

What is likely to happen and what he is to do, is the special concern of the practitioner.

Unfortunately, although septicemia and bacteremia are used in most of the medical writing to-day as interchangeable, they have not always been so used, nor are they consistently so used to-day. In the monograph, by Seitz, on the nature of infection in the 3rd edition of the large "Handbuch der pathogenen Mikroorganismen," published in 1929, there occurs, as in the older editions, the following paragraph: "The two conceptions, bacteremia and septicemia must be sharply distinguished. Bacteremia is purely a passive process in which the bacteria pour into the blood from a focus of infection. The blood serves only as a means of transportation for the bacteria. . . . When the exciting agent is not mechanically transported by the blood, but is actively multiplying in the blood current, then we speak of septicemia."

Such a conception has disadvantages. It takes no account of the great capillary fields which form an important part of the circulatory system, in the liver, spleen and bone marrow. It takes no account of the inability of the clinician to recognize the moment when a bacteremia, in the strict sense, passes over into a septicemia.

The distinction is definitely abandoned by Zinsser in his last edition of his Bacteriology. It is abandoned by most writers to-day. Bacteremia and septicemia are used as synonymous. Each year in the medical writings, articles appear which show this usage. In 1928, for example, in the *New England Journal of Medicine,* O'Brien reports a case of gonococcus septicemia with recovery, and a case of recovery from anthrax septicemia is reported from the *Presse médical.* Both instances might have been spoken of as examples of bacteremia.

Without a mental grasp of the sequence of happenings, when the body is infected with microörganisms, it is impossible to consider rationally, either prognosis or treatment, or this distinction attempted in the definition of certain bacteriologists, between septicemia and bacteremia.

There are two papers recording experiments on animals that I think well worth bringing to your attention even if you are already familiar with them.

In experiments, recorded in a paper by Hobo, the material used was fine India ink and the amount used from 2.5 to 3.0 c.c. The particles were the same size or smaller than staphylococci and the solution was injected into the ear veins of rabbits. The visible mucous membranes immediately became dark gray; at the end of ten minutes the color had disappeared. The animals were killed at periods varying from forty-five minutes to seventeen days after injection. At autopsy, the spleen, the bone marrow and the liver were found to be stained black. The other organs and tissues—the lungs, heart, brain, nervous tissue, gastro-intestinal tract and the kidneys were unstained. The microscopical examination showed in the early stages, particles of India ink in the star cells of Von Kupffer of the liver, the sinus endothelial cells and the pulp cells of the spleen and the endothelium of the capillaries of the bone marrow. In the capillary enlargement of the metaphyses of the long bones the particles were so thickly deposited that it was impossible to recognize the phagocytosis. In the kidneys, clumps of the pigment were to be seen here and there. Scattered through the various organs were mononuclear and polymorphonuclear leucocytes filled with black particles.

The precise and interesting experiments made by Wyssokowitsch over forty years ago are equally interesting. He used fresh cultures of moulds, saprophytic, pathogenic and non-pathogenic bacteria and experimented on dogs, rabbits and guinea-pigs. Relatively large quantities were injected into a vein; blood was then drawn at intervals of from ten minutes to twenty-four hours; cultures were

taken and the colonies counted. Enormous quantities of spores of moulds (aspergillus and pencillium) and saprophytic bacteria disappeared with astounding rapidity. Within three hours, millions of bacilli (Bacillus subtilis), injected intravenously, had completely disappeared. Bacteria, pathogenic for man, but harmless for the animals experimented on, disappeared in the same way. Thirty minutes after the injection of millions of cocci (Streptococcus pyogenes) into the circulation of a dog, only one colony was found in the blood culture; in one and a half hours the blood drawn was sterile. Even after injections, repeated each day for four days, of many millions of microörganisms (Micrococcus tetragenus) into the blood of rabbits, only six colonies had grown from the blood culture at the end of twenty-four hours. The disappearance of bacteria pathogenic for the animal, injected in small quantities, was equally rapid. In some instances it was complete: that is, the blood withdrawn from a rabbit, into the blood stream of which anthrax bacilli had been introduced, showed no organisms at the end of twenty-four hours. If larger doses were introduced, there was first a diminution, then an increase, until the bacteria became countless. In a rabbit injected with a large dose of anthrax bacilli, at the end of five minutes, only fifteen colonies were found in the withdrawn blood. At the end of two and a half hours there were no colonies: at the end of forty-six and a half hours, three colonies: at the end of seventy hours, fifty-six colonies: at the end of eighty-four hours, death followed with countless numbers of bacteria in the blood stream. The animal in which no organisms were found in the blood at the end of twenty-four hours, was killed. Cultures from the blood showed no growth; however forty-five colonies grew from a culture taken from the spleen and countless colonies from the liver. In animals that died after an injection of large doses of pathogenic organisms, autopsies regularly showed not only the liver, spleen and bone marrow, but all the tissues swarming with bacteria. The blood itself, in the larger vessels, contained but a small part of the myriads of microbes found throughout the body.

These two experiments show:

(1) That inert particles the size of bacteria rapidly disappear when introduced into the circulating blood and are found deposited, for the most part, in the liver, spleen and bone marrow.

(2) Nonpathogenic organisms are deposited in the same way and are destroyed.

(3) Pathogenic microörganisms in moderate doses are similarly destroyed, but, when more virulent, or in larger doses, multiply in these situations and invade the blood stream; first a few, finally in countless numbers.

The well known sequence of events in infection by spirochetes is also an aid in forming the picture of disseminated microparasites. The course of the disease in rat-bite fever, relapsing fever, syphilis and infective jaundice, show various phases: a latent period, sudden onset of severe symptoms, dissemination in the blood stream, and multiple transitory lesions and finally, disappearance of the microparasites and destructive lesions.

In experimental syphilis in the lower monkeys, even when there are no general manifestations, examination of the pulp of the spleen, the liver and the bone marrow after death, shows abundant treponemata even when the animal, during life, was apparently in perfect health.

Dissemination takes place from germs multiplying in these regions; the liver, the spleen and the bone marrow.

General infection by bacteria follows the same sequence.

By the time the general invasion occurs, the body has acquired a heightened capacity for destruction of the microparasites. The body's reaction to the invading microparasites has altered both locally and generally. The violent reaction, made evident clinically by fever, is an evidence of the destruction of the multitudes of microparasites. If the body cells hold in check the microbes, then a third stage is reached, with local lesions in the body and a more pro-

nounced reaction. The blood becomes powerfully bactericidal to the organisms causing infection, microörganisms entering the circulation are rapidly destroyed.

In most infections there is an initial lesion at the point of implantation indicating a local sensitization of the body and a heightened capacity on the part of the cells to react to the invading parasite.

There is a period of generalization when microörganisms are multiplying in the body and invading the blood stream, terminated by widespread destruction of the disseminated parasites.

There is a final period, after the microbes have disappeared from the blood, when local lesions appear from the settling out of the microbes at points of diminished resistance. These periods pass into one another and overlap one another, and are often made indistinct by reinfection and secondary infection.

When a lesion has become established in, or on, the walls of some portion of the circulatory system, during the final period, microörganisms may be poured into the circulating blood and time after time be destroyed.

A few clinical examples will illustrate these points.

Bacterial contamination of the blood stream is not infrequent following infections of the middle ear. The mastoid cells are in close proximity to the lateral sinus. It has long been recognized that infective thrombophlebitis occasionally follows infection here, either by direct involvement of the walls of the sinus or by extension to the sinus through one of the minute communicating veins and that such involvement is made evident clinically by chills and fever. Cultures taken during these periods very frequently show the presence of streptococci in the blood stream. The prompt ligation or removal of the infected vein stops the process and the patient recovers. Although large numbers of pathogenic streptococci have been poured into the blood, they disappear completely and the patient recovers if the

bacterial showers stop spontaneously or are cut off. The blood is bactericidal; the body cells destroy the microörganisms. I asked, not long ago, a distinguished and able otologist, who has ligated or excised the interjugular many scores of times, what had become of the hundreds of thousands of bacteria that had already poured into the blood stream before the infected vein was shut off, and he said he had not the slightest idea.

These patients present examples of bacteria disseminated in the blood stream by lesions in the vascular system, after the body has acquired an altered reaction, a heightened capacity for the destruction of the microörganisms causing the infection. If untreated, unless the primary lesion is very small, the bactericidal power of the blood gradually becomes less and less, the bacteria live longer in the blood, they gain a foothold in various parts of the body, myriads are finally poured into the blood stream and the patient succumbs.

A very different picture is seen in the following case report: A woman caught her wedding ring on some object and tore her finger badly. The wound was sutured. She entered the hospital with gangrene of the finger. Her temperature on admission was 99; the next morning the temperature was normal. The gangrenous finger was amputated, the skin closed except about the exit of a small drain. The first forty-eight hours after operation the temperature was 99½ to 100 3/5. At eight P. M. on the second day the temperature was 100 1/5. It then rose until her death at noon of the third day. Cultures from the heart's blood showed streptococcus hemolyticus.

In this instance, before the finger was amputated, streptococci had passed into the circulating blood. The virulence of the streptococci, or the lessened resistance was such, or, to put it in another way, the lessened capacity for bacterial destruction was such, that they gained foothold in spleen, liver and bone marrow, and invaded the blood stream.

Osteomyelitis furnishes another instructive clinical picture. Staphylococcus osteomyelitis is one of the lesions of staphylococcus septicemia. At the beginning of the disease the blood culture is usually positive. The sequence of happenings is often as follows: A child has a small furuncle or small infected wound. After several days or weeks, often when the initial lesion has healed, there is a violent general reaction, chill, fever, prostration. Staphylococci are found in the circulating blood and purulent foci appear in various parts of the body, especially in the expanded position of the shaft near the epiphyseal line where the experiments of Hobo show particles of carbon were deposited in the largest amount.

One sees patients in whom osteomyelitis is only one of many purulent lesions found in the body, others in whom only a single bone is involved, and still others in whom two or more bones are involved. Moreover, when the microörganisms are arrested and start to grow, they may set up a marked lesion in one place and a very slight one in another. Finally the circulating cocci disappear from the blood and localized lesions occur with death of tissue. I give briefly a clinical history: A boy two years old was operated on for a painful swelling over his third metacarpal bone. He presented the usual picture of chronic osteomyelitis. His history was as follows: Eight months before he had had a phlegmon following an infected wound on his forehead. After this he became very ill with signs of broncho-pneumonia, which was followed by an abscess on the back of his hand in the region of the present swelling and in the lateral thoracic region. A positive blood culture showed Staphylococcus aureus. The abscess on his hand and the one in the lateral thoracic region were incised and cultures of Staphylococcus aureus were obtained. He developed evidences of pericarditis; 18 c.c. of purulent fluid growing staphylococci were aspirated; ascites appeared. Aspiration showed straw colored serum; cultures were negative. The patient had had an acute staphylococcus sepsis with an abscess in the subcutaneous tissue, purulent pericarditis and osteomyelitis. At opera-

tion eight months after the onset the usual signs of chronic staphylococcus osteomyelitis were present. The various foci in the body had healed; only where the lodged cocci had caused gross tissue death did infection persist. After removal of the necrotic tissue, the patient recovered. Seen nine months later he was in perfect health, fat and rosy. A year later a staphylococcus abscess was opened in the shoulder region and after several weeks healed. At present he has a painful area over the middle shaft of the humerus. The X-ray shows thickening of the periosteum at this point. He has a low grade residual infection of the humerus.

The illness of the King of England was given wide publicity and furnishes another clinical picture. He suffered from a streptococcic septicemia which finally localized in the base of the right lung and which was followed by empyema. A rib was resected and he has slowly recovered. To quote from the London letter in the *Journal of the American Medical Association*: "In the first phase of this severe illness, gradual in its onset, there were noted general infection with imperfect localization; little or no cough and only one small patch of pleuritic friction; a blood culture positive for streptococcus, and an irritative state of the nervous system which produced profound distress and a sense of illness. Toward the end of the first phase came an accentuation of pleuritic friction, which extended into the diaphragm. The second phase was one of increasing toxemia with dusky appearance, dry cracked tongue, periods of delirium and exhaustion—in short, a clinical picture resembling that of severe typhoid in the third and fourth week, but with the added anxiety of attacks of dyspnea and cyanosis due to strain on the heart. With the next phase came an abatement of fever and some evidence of localization. The blood culture was now negative, and toxemia was less with delirium subsiding. The localization in the right lung did not result at this stage in effusion as was shown by puncture and excellent roentgenograms. A few days later the temperature rose rather abruptly to a higher level, and on December 12 there was

evidence at the extreme right base of an effusion which had begun between the lung and the diaphragm. Drainage by means of rib resection was performed on the same day under general anesthesia—gas, oxygen and ether. The prominent organism in the empyema was found to be identical with the streptococcus found in the blood."

It is unnecessary to give further examples. Our conception of septicemia, built up by clinical observations made when pathogenic bacteria are detected in the circulating blood, must take into account all these cases. Examination of the contaminated blood gives but small indication of the complex phenomena taking place. The number of microbes discovered may be but a portion of the myriads in the body or may only indicate the passage of hundreds of thousands on their way to destruction.

The forecast of the patient's fate can only be made by considerating the entire clinical history in connection with the blood culture.

It is not necessary to be hopeless when pathogenic germs are found in the blood current. In many patients the germs are poured into blood powerfully bactericidal. In many the germs are found in early stages of disseminated infection, which will pass on to localization and recovery.

The outcome in any given case depends on the number and virulence of the bacteria, the resistance of the host and the character of the focal lesions.

An attempt might be made to base an opinion on the number of colonies found in the blood cultures. It is self-evident that a single colony found on a single plate must have a different significance from hundreds of colonies found on every plate, but only by repeated examinations and a comparison of a number of similar cases can an indication be given regarding prognosis, and whether or not the bacteria are pouring into the blood stream with bactericidal properties, or are multiplying in the liver, spleen and bone marrow.

These facts also furnish a guide for intelligent treatment.

Obviously, proper nursing, rest, administration of fluids and careful observation are measures applicable to all severe illness. I shall only refer to measures especially called for in septicemia.

The ligation and excision of a vein, the seat of suppurative thrombophlebitis, if done early, is a most satisfactory measure. Here, as we have said, the bacteria are passing into a blood stream which has bactericidal properties, shutting off further contamination.

Unfortunately there is only one region in the body where the anatomical relation and arrangement of the infected area and the tributary veins offer suitable conditions for surgical interference. I refer to the mastoid cells, lateral sinus and jugular vein.

There were ten recoveries and three deaths in a series reported from the otological service of St. Luke's Hospital. In the ten patients that recovered, eight had the jugular ligated immediately after the streptococci were discovered in the blood. There were two patients that recovered without ligation; in one there was only a single culture showing one colony. Two were instances of severe blood stream infection with hemolytic streptococci. Both recovered after ligation.

A similar condition occasionally occurs in an area of infection in an extremity. Amputation above the infected area is followed by complete and startling recovery. An observation of Dehelly is interesting. He amputated the leg of a fireman for an infected compound fracture. The patient continued ill and repeated examination showed hemolytic streptococci in the blood. He reamputated through the thigh. The patient promptly recovered. Thereafter the blood cultures were negative. A most painstaking examination of the amputated stump showed suppurative thrombophlebitis of a single vein.

In puerperal sepsis the effort to remove the uterus with the ovarian veins which at times show suppurative thrombophlebitis has met with little success. In most instances we are dealing with a suppurative thrombophlebitis occurring in the early stages of dissemination.

In infection of the lips and face with suppurative thrombophlebitis, both the jugular and facial have been ligated to prevent dissemination. Lexer reported a case of carbuncle of the cheek in which there was a purulent thrombophlebitis of the facial and general infection and in which he was able to avoid further metastases and cure the patient by ligation of the jugular.

Two years ago Roeder reported a case in which he records ligation of the angular vein in an infection of the upper lip. The vein was not thrombosed, culture of its walls and lumen were negative. There were no positive blood cultures. The measure was said to be prophylactic. There is nothing to indicate that the patient would not have recovered without ligation.

Attempts have also been made to excise the initial lesion under the impression that the microörganisms are finding their way into the circulation from this primary focus. Such treatment is nearly always unsuccessful, often harmful. The microörganisms found in the blood stream are usually the organisms multiplying far from the site of inoculation.

The study of the treatment of anthrax serves as an illustration. Twenty-four years ago the text books of surgery advised excision of the skin lesion of anthrax, followed by the application of caustics, strong antiseptics, or the actual cautery. Small local lesions recovered after this treatment but the results were not as good as when the lesions were protected from injury, the parts kept at rest, and secondary infection avoided. Apparently in some instances the bacilli seem to have been disseminated by the operation. During the last ten years a powerful anti-anthrax serum has been available and has been used with

and without excision. There is a growing recognition that the disease is often local and shut in by a barrier of cells; that excision may break through this barrier and disseminate the bacilli and may cut out a focus perhaps furnishing antibacterial substances; and that, if the microörganisms are widespread, as is often the case, local excision is useless.

The following English statistics of the Ministry of Health published in 1921 are interesting:

	Cases	Deaths	Mort.P.C.
Serum alone	200	8	4.
Excision only	397	44	11.1
Excision and serum.....	174	25	14.4
No special treatment.....	29	14	28.3

The secondary lesions that appear early in dissemination are the result of bacteria deposited in special sensitized areas or in areas of least resistance or areas that offer anatomical arrangement unusually favorable for the growth of the microörganisms.

Incision or evacuation of exudate in these instances can have only an indirect effect on the microörganisms in the circulation. Such foci are the result of dissemination, not the cause.

It is a common observation and one confirmed by experience that mechanical interference, during the early stage of infection should be confined to the simplest measures, such as aspiration and simple incision to afford drainage and thus avoid unfavorable pressure from the exudate. It is only after the bacteria have disappeared from the circulation and the local focus is well circumscribed, that incision and drainage show satisfactory results. This was well shown in the streptococcus infections that occurred in several of the camps during 1918. Extensive operation on the chest when there was a thin turbid exudate in the pleura and streptococci in the blood showed high mortality. These patients were suffering from streptococcus septicemia as the multiple lesions at autopsy showed.

Aside from these purely surgical measures which can only be applied in special cases with localized lesions, there are certain general measures in use to-day, such as bacteriotherapy, serotherapy, chemotherapy and the transfusion of blood. My own experience in these fields is limited and my attitude sceptical. I have seen no such convincing evidence of the efficacy of any measure suggested as to feel justified in advocating it whole-heartedly. I shall review therefore a few of the reports published during the last two years. In 1928, Cadham reported the history of eighteen patients with septicemia treated with repeated inoculations of a homologous immune animal serum and repeated transfusions of human serum, with sixteen recoveries and two deaths. The next year Prof. Vincent, of Paris, reported very favorably on a new anti-streptococcus serum, and Dujol, in the same year, reported recovery from a staphylococcus septicemia, after injection of an auto-vaccine prepared from fluid containing pus and staphylococci withdrawn from the pericardium. In January of this year Rosher reported in the *Lancet,* four cases of streptococcus septicemia in which Streptococcus hemolyticus was isolated from the blood and in which complete recovery followed treatment with scarlet fever streptococcal antitoxin, and several weeks before Burt-White reported twenty-seven cases with a morality of 29.6 per cent treated in the same way.

I shall offer, after the discussion of the introduction of chemicals into the circulation, reasons for not accepting too readily, the conclusions drawn from these favorable reports.

During the war, Anderson and Richardson, in the *British Journal of Surgery* of 1917 and 1918, reported favorable results from intravenous injection of eusol, one of the preparations of hypochlorite of soda. During the last twelve years similar reports have followed the introduction, intravenously, of other antiseptics; perhaps the best known is mercurochrome. About the time of its introduc-

tion wide publicity was given to the astonishing, or as they were then described, truly wonderful results.

It is well known that valuable as antiseptic solutions are in preventing secondary infection, in sterilizing the skin, instruments etc., there are reasons why their use, when bacteria have become established and are multiplying in the body, is unsatisfactory:

First, any chemical substance which kills bacteria when brought in contact with them outside the body acts by some sort of reaction between the chemical and the protoplasm of the bacterium, an analogy existing between the ordinary chemical reaction between two reagents and the reaction between the antiseptic and the bacterium. The reaction is by no means simple, nor is it the same for the different chemicals, but in all cases it is connected in some way with damage to the surface of the bacterium so that it cannot carry on the normal exchange on which its life depends.

Second, the antiseptic has a tendency to combine with substances other than the bacterium with which it comes in contact. Therefore, the living cells, the various chemical ingredients and albumins in the body unite with the antiseptic.

Third, the antiseptic, just as any chemical, is used up and rendered inert by combining with the various substances with which it comes in contact.

Fourth, to be effective, the antiseptic must come in contact with the body of the bacterium. That is, a germ inside a cell is not killed by the antiseptic if the chemical combining property on which its activity depends is quenched before it reaches the body of the germ.

Fifth, the active cells on which the destruction of the bacteria depend are more delicate than the bacterium. Their surface condition, on which their normal function depends, is even more liable to be affected in a harmful manner than the surface of the bacterium by chemical solutions; the living cells, even if not killed, may be damaged.

All these considerations make it improbable that any antiseptic applied with the idea that it is a simple germicide will be effectual. Notwithstanding these facts, which have been brought out by careful and patient studies, very favorable results continue to be reported from the intravenous injection of antiseptics. In 1930, Harris reported in the *United States Veterans Bureau Medical Bulletin*, that a patient recovered after nine intravenous injections of mercurochrome. The patient was suffering from Streptococcus viridans septicemia. The same year Veillon reported in the *Bulletin de Soc. méd. des hôpitaux*, recovery from meningococcus septicemia after three injections of acriflavine hydrochloride solution. In August, 1929, Bernstine reported sixteen cases of blood stream infection in which metaphen, introduced intravenously, had a beneficial effect. All were cases of puerperal septicemia.

The difficulty in interpreting these results is that each case quoted must be carefully studied and analyzed by itself. For example, the cases reported by Dujol as recovering after administration of autogenous vaccine are similar to the one I have reported as recovering without vaccine. Did the patient reported by Harris recover on account of the nine injections of mercurochrome or in spite of it? One case report does not furnish proof of the efficacy of a given form of treatment.

As I have said, there has been as yet, no general recognition of the curative value of any of these measures, yet one cannot dismiss without comment the evidence of sudden and rapid recovery presented in many of the case reports. In certain instances there is evidence of severe disturbances after the introduction of chemicals and after the introduction of serum. Is some form of protein shock a common factor, either from the serum introduced or from damage to body cells by the chemicals? Is the protective mechanism of the body stimulated or reactivated? As one watches the case reports year after year, one is surprised by the similarity of the results from widely different measures and widely different chemicals. Obviously it is diffi-

cult to estimate the value of treatment where such different conditions are grouped together under a single term, and where the sporadic nature of the disease makes the numbers that come under the observation of one observer so limited.

In regard to transfusion of whole blood, there does seem to be a general agreement that it is beneficial, at least temporarily, especially in subacute and chronic cases. Repeated small transfusions seem to be more effective than a single large one. The color improves, the feeling of well being returns, the appetite improves; it seems difficult not to believe that it is helpful in aiding the body to combat disseminated infection.

I have endeavored, in this cursory talk, to interest you in the phases in the course of infection, and to bring to your attention the necessity of bringing a critical attitude (founded on a knowledge of, one might say, the vagaries of septicemia) toward the various measures that are enthusiastically advocated, often perhaps, to the detriment of the patient.

There seems to be a widespread, although erroneous impression that a patient in whose blood streptococci or staphylococci are found is doomed to death. Therefore recovery following any form of treatment is set down as extraordinary and the agent used extolled.

COMPLICATIONS INCIDENT TO INACCURATE OR DELAYED DIAGNOSIS IN SUPPURATION OF THE LUNGS AND PLEURA*

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When your medical secretary invited me to discuss the complications incident to inaccurate or delayed diagnosis in suppuration of the lungs and pleura, I was much gratified at the prospect of discussing this interesting subject, but on reading the invitation further it specified "from the standpoint of the practitioner rather than that of the surgeon," and my enthusiasm waned somewhat, as I doubted if I could do the subject justice from that aspect, as most of my work is surgical and all of our chest operations at the Walter Reed Hospital are based on the combined findings of the internist, the radiologist, the bronchoscopist and the surgeon. However, on reconsideration I felt that we had learned much that might be of interest to the practitioner, by deductions made from our mistakes and the mistakes of others in the handling of over 400 cases of non-tuberculous empyema, 90 odd cases of tuberculous empyema, and a reasonable number of lung abscesses and bronchiectasis.

Any discussion of the complications incident to suppuration of the lungs and pleura would be incomplete, especially from the practitioner's point of view, without some reference to the acute processes that antedate these complications. The same also applies, even if only superficially, to treatment, as proper treatment of the acute processes is still our greatest asset in prevention of the chronic ones and the complications incident to these.

Empyema has always been an important question for the army surgeon, and if by any effort we can prevent a

*Delivered October 24, 1930.

catastrophy like that of 1918 when mortality in our camps ranged from thirty-two to eighty-four per cent, we will have achieved something worth while. In addition to this high mortality there were 970 cases of empyema with discharging sinus at the time of the Armistice, many of whom succumbed later at their homes, while others were passed along from one hospital to another for years, when an early accurate diagnosis with conservative surgery might have solved the problem in a large percentage. From an epidemiological aspect we gained little from our experience in 1918, but from a medical and surgical point of view, it is not so discouraging.

Now, as to who was responsible for the high mortality and complications, whether the surgeons operated too early or too late, or whether the practitioner failed to differentiate between empyema with active pneumonia that should not have been operated upon, or the one that should have been, is not the province of this discussion tonight. What I hope to give you are certain proven facts based on the findings of the empyema commission during the war and those of individuals throughout the country since the war *as to what should not be done,* which, if applied in future epidemics, should lower the mortality in empyema to less than 10 per cent in adults and reduce chronicity and complications to a minimum.

It is questionable if there is any one factor as important in the prevention of the complications incident to suppuration of the lungs and pleura as an early accurate diagnosis, especially when it is based on a carefully taken history, a thorough physical examination and proper analysis of the data obtained.

It has never been definitely determined why there was so much difficulty in obtaining an early and accurate diagnosis in the flu empyemas in many camps during the epidemic. The text book picture was supposed to have led to confusion, and the symptoms that were most constant could be counted on three fingers; that is, dullness on per-

cussion, diminished or absent tactile fremitus and diminished breath sounds. These symptoms, plus the x-ray and the aspirating needle, were responsible for more accurate diagnosis than all other symptoms. This was especially true when aspiration was done at the point of greatest dullness, using due precaution to avoid liver dullness, and when negative in the face of other positive findings was repeated and repeated in many directions. The x-ray was of inestimable value in differentiating between pleural fluid and consolidation as well as in determining the height of the diaphragm on the side to be aspirated. Such symptoms as pain, irregular temperature, chills, leukocytosis, intercostal bulging, cardiac and tracheal displacement, change in fluid level, change in the cardio-hepatic angle, and change in the costo-phrenic angle were present in a certain percentage of cases, but they were not constant, for often where the fluid was encapsulated high up or interlobular, the costo-phrenic angle was not obliterated, the cardio-hepatic angle was not always changed, the fluid level did not shift, the trachea and heart were not displaced, and intercostal bulging was not present.

Some of the confusion has been attributed to the fact that in the streptococcus pneumonia, the leukocytosis irregular temperature and chills are often due to the persistence of intrapulmonary streptococcus infection.

Acute empyema in the first stage, with a purulent exudate present on aspiration, results from an infectious process in which the first stage is a pneumonia with a small sero-fibrinous exudate, and the second stage a pneumonia with a sero-purulent exudate. The mechanical effect of the exudate in the pleural cavity causes compression of the affected lung, pressure on the mediastinum and heart, as well as pressure of the unaffected lung. Symptoms are influenced by the location and amount of exudate; that is, it depends whether it is interlobular in the spinal gutter or free in the pleural cavity, the latter condition being influenced by the character of the exudate, as a serous exudate is usually free in the cavity, while the purulent

exudate is nearly always encapsulated, due to deposit of fibrin at the periphery. The actual primary location of the empyema is supposed to depend on rupture of a small subpleural abscess.

In acute empyema it is imperative that we determine whether the etiological factor is the hemolytic streptococcus, the pneumococcus, or the tubercle bacillus before surgical measures are undertaken, because the hemolytic streptococcus tends to produce a low vital capacity. The low vital capacity present in hemolytic streptococcus infection makes a free opening in the chest dangerous in the early stages of the disease, as the sudden change from normal negative intrathoracic pressure to abnormal atmospheric pressure may prove fatal from collapse and mediastinal displacement, as this type of case in the early stages is often cyanotic, suffering from air hunger to such an extent that they have difficulty in receiving their tidal air and, as pointed out by Graham, even a minute opening in the chest may prove fatal, as the pneumonic process has already infringed on the air space and reduced it to a minimum by extensive occlusion of the bronchioles, both by actual plugging with exudate and by edema. Owing to the fact that the exudate often appears as early as the fifth day in this form, the respiratory and circulatory embarrassment may have to be relieved by aspiration until the pneumonia has subsided and the fluid has become purulent and rich in fibrin, when one of the recognized negative pressure operations can be done with practically no mortality. On the other hand, with an early open thoracotomy in such cases, fluid escapes, air rushes in, lung collapses, and death often ensues from mediastinal disturbances or toxemia incident to the still active pneumonia.

The condition is different when we are dealing with empyema due to the pneumococcus, for in this type the pleural fluid is rich in fibrin, insures early adhesion of the pleural surface which stabilizes the intrathoracic viscera, important in preventing post-operative lung collapse and mediastinal disturbance. Usually when the exudate

is recognized, the active pneumonia has subsided, and we are dealing with a condition we hope for when we delay operation in a hemolytic streptococcus pleuritis and when we wait for the fluid to become purulent, and for this reason it is important that we know the bacteriology of each case. Before the late war an early thoracotomy was used rather generally and often with good results when used on the type due to the pneumococcus, but when used in the early stages of the hemolytic streptococcus type, the results were often as disastrous as they were in our camps in 1918 when the mortality was prohibitive, the complications legion, and many of the 970 cases with discharging sinuses at the Armistice were of this type and treated by an early open thoracotomy.

In tuberculous empyema the general practitioner can, by a carefully taken history and a thorough physical examination, do much to simplify the diagnosis. It is, as you know, a form of suppurative pleuritis, in which the tubercle bacillus is the only etiological factor, but often invasion by other organisms results in mixed infection. The greatest tragedy that can befall the closed tuberculous empyema patient with sterile exudate in the chest cavity is to subject him to an open thoracotomy operation, which always means mixed infection and much unnecessary radical surgery that these patients, already handicapped by tuberculosis of one or both lungs, should not be compelled to undergo. However, a mixed infection might be unavoidable when it is accidental from aspiration or when metastatic. In diagnosis of tuberculous empyema history is most important; that is, a history of previous pleurisy, tuberculosis of other organs, peritoneum, or lungs adds weight to the diagnosis when a sterile fluid is found in the chest. The undrained sterile purulent exudate in the pleural cavity with fever and low leukocyte count is almost always tuberculous. Demonstration of the tubercle bacillus in the exudate is possible in many cases, and animal inoculation, even if slow, is very satisfactory. In open cases pathologic examination of the excised pleura is enlightening.

I will now show the slides of ten cases of tuberculous empyema, which should help demonstrate the tragedies incident to inaccurate or delayed diagnosis in tuberculous empyema. They represent a five year follow-up in ten cases that were either bacteriologically or guinea pig positive for the tubercle bacillus.

Group No. 1—Five cases with sterile purulent exudate in the closed chest cavity and no active tuberculosis in the so-called good lung. No open thoracotomy or mixed infection. Sputum negative for tuberculosis. Exudate positive for tuberculosis.

Group No. 2—Five cases. All five had an open thoracotomy with mixed infection and multiple operations before admission to the Walter Reed General Hospital. No active tuberculosis in the so-called good lung. Sputum negative for tuberculosis. Excised pleura positive for tuberculosis.

All of the cases in Group No. 1 are living and in good physical condition at the end of five years.

Of the cases in Group No. 2, 60 per cent are dead at the end of five years, many showing beginning loss of weight at the end of three years. The remaining 40 per cent now living show, as you can see, marked emaciation.

It was my intention to show the slides of twenty of these cases, but found it would be impossible to present that number in the brief time allotted. However, the slides of these cases should be sufficient to demonstrate the tragedies incident to open thoracotomy in tuberculous empyema cases. Time will not permit of my going into detail relative to mixed infection in closed tuberculous empyema cases, with mixed infection of metastatic or accidental origin, other than to say that they should be sterilized, if possible, by the closed or negative pressure method, and their cavities should then be obliterated by extrapleural thoracoplasty.

As to the prognosis in tuberculous empyema in patients with active tuberculosis in the so-called good lung, it is

difficult to determine, as everything depends on the progress of the tuberculous process, but usually those not subjected to an open thoracotomy live longer.

The treatment of chronic non-tuberculous empyema is more or less standardized and the mortality has been reduced to less than 9 per cent by the many-stage open method of treatment, as shown by the lantern slides, which are self-explanatory.

A word as to the complications incident to the chronic form—I would like to add that many are profoundly septic, depressed mentally and physically, fingers clubbed, joints swollen, heart, kidney and other complications suggestive of amyloid changes incident to prolonged suppuration.

The usual picture is that of one or more cavities with thickened sclerotic wall over a lung that may be collapsed. The intercostal spaces are narrower, the ribs are more vertical, the thorax is retracted, and chest wall is often traversed by sinuses. These slides demonstrate the open many-stage operation for chronic empyema and the results obtained.

Acute Lung Abscess—In this condition, as in acute empyema, the proper treatment of the acute is still our greatest asset in prevention of the chronic, and this is especially important since the surgery of the chronic lung abscess is anything but satisfactory, and the mortality is still high, except in those cases where a single abscess is located superficially, and in selected cases suitable for lobectomy.

Before discussing the treatment of acute lung abscess, I want to mention some outstanding points in pathology and symptomatology which might be helpful to the practitioner in diagnosis and the surgeon in treatment. Acute lung abscess is seldom primary, usually being associated with other chest conditions. About one per cent are due to foreign bodies. Prior to the war we considered pneumonia as the prime factor in the etiology of acute lung abscess. This we now know to be incorrect, and that most

of them are post-operative, following chiefly abdominal and tonsil operations, though they may occur after any operation, whether performed in a clean or septic field. They may be parenchymatous or endo-bronchial, single or multiple. The single abscess is usually in the lower lobe and more common in the right lung than the left. In pneumonia the abscess begins in the area of consolidation, which is also true of abscess resulting from septic embolism, the emboli first producing an infarct.

In acute lung abscess there is an area of coagulation, necrosis, and partial liquefaction that is surrounded by an area of intensive hyperemia and edema. Later the cavity may be filled with pus that varies in odor and consistency. At this stage there may be no well defined wall, the cavity being surrounded only by a zone of necrotic lung tissue, which is surrounded by a zone of hyperemia and congestion. When the abscess becomes chronic it is surrounded by a well defined connective tissue wall, which is penetrated by bronchi. Whether lung abscesses are embolic or respiratory in origin has never been definitely determined. If the abscess is post-operative the symptoms may appear within twenty-four hours, or they may not develop for a week. Symptoms may appear early or late, usually beginning with fever, cough, profuse expectoration and pain if the abscess is close to the pleural aspect. The fever is intermittent with chills; there is sweating and a high leukocyte count. The condition is often mistaken for pneumonia before the sudden expectoration of a large quantity of sputum, and sometimes difficult to differentiate from an encapsulated empyema that connects with a bronchus. The real physical signs of lung abscess may be present only after the cavity is evacuated; that is, tympany on percussion, whispering pectoriloquy and amphoric breathing. The abscess may terminate by rupture into a bronchus, or rupture into the pleural cavity and cause an empyema, or it may rupture into both pleural cavity and bronchus. Inadequate drainage may lead to chronicity, general sepsis, hemorrhage, gangrene, brain abscess and other complications.

The proper early application of postural drainage will greatly increase our present percentage of cures. This cannot be carried out by a rotating personnel, because a sufficient number of postural changes necessary to keep the patient free from that sense of irritation and fullness that precipitates coughing spells must be made, which requires a close observation of the patient.

For the proper application of postural drainage an early diagnosis is imperative and should start at once when drainage is established between the abscess and the bronchial tree while the lung abscess is still amenable to collapse and before a definite connective tissue wall prevents collapse. The slides shown here represent the results obtained when postural treatment was instituted early; when I say early I mean immediately after the patient expelled a large amount of sputum during the act of coughing, especially when this expulsion of a large volume of sputum is preceded by a period of respiratory symptoms with fever, cough and leukocytosis, and where an encapsulated empyema secondary to a pneumonia can be eliminated.

The treatment of chronic lung abscess has been sufficiently illustrated by the lantern slides. The prevention of chronicity by proper treatment of the acute has been purposely emphasized.

ACUTE INFECTIONS OF THE GALL BLADDER AND BILIARY TRACT*

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Gall bladder inflammation and gall stone disease has been recognized for centuries. But the clinical picture and diagnosis of these conditions was not recognized until the last century.

A careful history and a thorough physical examination are the most reliable and the most practical of the various diagnostic measures applied to the biliary tract. The reasons for this are that the biliary tract and the factors that produce its diseases are more amenable to analysis and accurate localization than any other tract unless it be the urological. The factors of gall bladder concentration, inflammation and obstruction, in relation to the part of the gall bladder or bile passages in which one or more of these factors is active determine the clinical picture. As an example: If during a pregnancy the cholesterol, which is in excess in the bile, becomes concentrated in the gall bladder beyond the saturation point, a pure cholesterol stone is precipitated and may remain as a silent stone for months or years. However, if it becomes impacted in the cystic duct, the factor of obstruction is added and a hydrops of the gall bladder will result if the factor of obstruction persists in that part of the gall bladder. If in addition the factor of infection and inflammation is added to the previously existing two factors in the same part of the tract, an empyema will result, giving a different clinical picture and demanding a more urgent therapy.

Permutations and combinations of these three factors in the several parts of the biliary tract, make the study of its diseases a most fascinating one. I have noted for

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many years that if the subject is presented in this way to the medical student, his attention is immediately focused and instead of a jumble of unintelligible Greek terms like cholangitis, cholecyst-duodenostomy, the subject becomes one of the most interesting and logical in abdominal diagnosis.

Therefore again I wish to repeat that a careful history and physical examination are the most important features of the diagnosis of biliary tract disease. In recent years, however, the experimental work of Rous, Graham, Whitaker and others, in demonstrating the concentrating activities of the normal gall bladder and applying the use of halogen derivatives to the roentgenographic demonstration of the gall bladder, has added a most valuable method to accurate and early diagnosis of gall bladder disease, and in many atypical or obscure digestive disturbances, gall bladder disease is shown to be the only lesion present. I should say that in the majority of well-established gall stone or gall bladder diseases the cholecystogram is not necessary to the diagnosis—certainly before its introduction by Graham in 1924, we were able to make correct diagnoses in over 75 per cent of the cases—but in the obscure case and in the field of differential diagnosis of upper abdominal lesions, cholecystography is invaluable, and when possible should be used.

I have digressed from the topic of my paper purposely to lead up to the statement that the great majority of acute gall bladder and bile tract inflammations are super-imposed upon chronic lesions and with few exceptions after gall stones have formed. That is, the acute phase usually appears long after the patient has been the victim of colic and interval digestive disturbances such as bloating, belching, and a sense of oppression in the upper abdomen.

How often in the presence of this background, when a patient comes down with a severe colic and a superimposed acute inflammation he is told by his physician that he is suffering from "acute indigestion." This is no news to the

patient, for he knows only too well that he is suffering and that his indigestion is acute. But that he should be made to feel that his trouble is merely a symptom and that the physician should, on that assumption fail to note an increasing tenderness and rigidity, and allow his patient to go on to a peritonitis or cholangitis is one of the unpardonable sins of our profession. If in the early stages of an acute cholecystitis, the process is recognized, and proper treatment is applied, many cases will subside and surgery deferred to a more favorable period of the disease.

In presenting the subject of acute cholecystitis, I have utilized the material and the experience of a general hospital, to which patients are sent by the practicing physician and cared for by members of the surgical staff.

The study comprises the data from 160 cases of acute cholecystitis operated upon at the Presbyterian Hospital during the period of 16 years since the establishment of our Unit Record System. These are unselected consecutive cases coming in on a general surgical service. During the same period we have operated upon 1600 patients with chronic cholecystitis with and without stones. That is, the acute cases comprise about nine per cent of the inflammatory diseases of the gall bladder. This corresponds fairly accurately with the figures of other clinics. Thus Graham reports 7.6 per cent of acute cholecystitis in their gall bladder series at the Barnes Hospital in St. Louis.

Acute inflammation of the gall bladder reveals itself in many forms, depending upon the factors of obstruction and inflammation and the part of the gall bladder involved—as previously stated. To make too many subdivisions such as acute catarrhal, suppurative, gangrenous cholecystitis is a mistake. As we understand acute cholecystitis it is a lesion showing edema, polymorphonuclear infiltration of the various layers of the wall of the viscus, with or without stones, with or without preceding chronic inflammation. The lumen may or may not contain pus. There may or may not be a pericholecystitis. As in other acute in-

fections, the onset is usually sudden, at times with a chill. Usually there is nausea and frequently vomiting. Pain is almost invariably present, usually of a continuous aching type, though there may be, and often is, the associated very severe gall stone colic. The temperature is usually around 101 degrees. Physical examination usually reveals a tender mass in the right upper quadrant, and with peritoneal involvement, involuntary upper right rectus rigidity appears. If the cystic duct obstruction by stone or edema of the gall bladder persists, if it is distensible it becomes larger, more tender, more easily palpable. If it has been the seat of long standing chronic inflammation, it is more liable to pericholecystitis and omental adhesions, with less easily palpable mass. Slow perforation with subhepatic abscess is more liable to occur. In the non-calculous acute gall bladder, or one that has not been the seat of previous chronic inflammation, perforation when it occurs results in a rapidly spreading peritonitis. Fortunately this is a rare occurrence.

It is the symptoms of pain and interval digestive disturbances that cause confusion in upper abdominal diagnosis. Pain in gall bladder and biliary tract disease is more frequent in women; it usually comes at irregular intervals, unrelated or without any regular chronological relation to food intake, as is the case in gastric and especially in duodenal ulcer. It is not relieved by taking food. It is more commonly associated either immediately or in the interval, with a sense of bloating and actual belching. The pain usually radiates to the scapula or to the back; it frequently ceases abruptly. Nausea and vomiting are usually limited to the onset of the pain and do not bring relief as regularly as in ulcer. The vomitus, as a rule, is greenish and is not characterized by fresh or old blood as is so frequently the case in ulcer or gastric cancer.

The interval digestive disturbances of biliary tract disease are usually described as a sense of bloating and of fulness or a lump in the epigastrium or left upper quadrant, whereas in ulcer or carcinoma of the stomach the

epigastric distress is of a burning, gnawing type. In the former, belching of volumes of gas is typical; in the latter, eructations are usually associated with the bringing up of sour, acrid or burning stomach contents. The pain in pancreatic disease, rarely unassociated with gall bladder or biliary tract disease, is epigastric and usually radiates directly through to the lower dorsal or upper lumbar region. Such pain, with tenderness extending across the upper abdomen, indicates a pancreatitis, and is frequently found in long-standing biliary tract disease.

A right-sided renal infection, especially a pyonephrosis, may be confused with an empyema of the gall bladder. In such cases there is usually a history of pain radiating from the back down to the groin or genitals and a history of cloudy urine. The finding of a large, tender mass in the right upper quadrant may be very suggestive of a large tender gall bladder, but a pyonephrosis usually can be felt by bimanual palpitation; tenderness is usually marked in the right costovertebral angle over the 11th and 12th ribs. Urinalysis, if repeated, usually shows gross or microscopical evidence of pus or blood in the urine, or if none is present, cystoscopy will reveal a non-functioning right ureter.

A right subhepatic abscess may be difficult to differentiate from an acute cholecystitis or empyema of the gall bladder. Such an abscess may be secondary to a duodenal, appendiceal, perinephritic, or gall bladder lesion. Here the history is most helpful in determining the initial focus. Too much time should not be spent, however, in determining the primary focus with the signs all pointing to an acute abscess in the right upper quadrant. The indications for drainage, if they are clear, should be met promptly.

There are, however, certain lesions which are at times readily confused with acute gall bladder and biliary tract disease and which in every way contraindicate surgical therapy. These are pneumonia, acute pleurisy, certain

heart lesions, catarrhal or infectious jaundice, certain lesions of the liver, such as acute yellow atrophy, cirrhosis, and syphilitic hepatitis, and rarely herpes zoster and Pott's disease. The mere enumeration of these varied lesions emphasizes the importance of a careful history and a thorough general physical examination. The severe right-sided pain of a diaphragmatic pleurisy, with or without a pneumonitis, may simulate the right upper quadrant pain of an acute cholecystitis. The history of an associated upper respiratory tract infection, the finding of a concurrent coryza, tonsillitis and bronchitis, with the presence of rapid, restricted respirations, a to-and-fro pleural friction rub on the lower right chest, with or without signs of beginning consolidation, should always give pause to any thought of surgery. Such a patient must be watched, and even if an acute gall bladder condition is present should be treated conservatively. In pneumonia the rapid, grunting respiration, working of the alae nasi, and high leukocytosis, even when chest signs are absent, are warning signs pointing to a chest lesion and demand a watchful attitude.

In certain heart lesions associated with acute cardiac decompensation, pain and tenderness in the right upper quadrant may simulate an acute liver or gall bladder infection. But the history of rheumatism, long-standing heart disease, the presence of orthopnea, dyspnea, cyanosis, rapid tumultuous heart action, cardiac enlargement, a tender, enlarged liver, leave no excuse for a mistaken diagnosis.

In addition to the tender liver associated with the decompensated heart, certain patients with coronary thrombosis complain of great epigastric and upper abdominal pain together with tenderness in these regions. They may also show a leukocytosis. These patients, however, as a rule, give a history of previous heart disease, are usually dyspneic, with rapid and sometimes irregular pulse, and frequently complain of precordial pain as well as epigastric pain. The sign which is pathognomonic of coronary thrombosis and distinguishes it entirely from acute lesions

of the upper abdomen is the appearance of the friction rub over the pericardium. The to-and-fro "shuffle" should be looked for in every case giving a history of previous heart disease. It may be transient, so that repeated examinations are necessary to detect it.

Catarrhal and infectious jaundice usually occur in the young adult and come on without previous history of similar attacks; pain is, as a rule, not associated. In certain forms of infectious jaundice merging into mild acute yellow atrophy, a tender liver with deepening jaundice may be present. In such cases the decrease in size of the liver, with an increase in jaundice and appearance of mental dulness or psychosis, points to a non-surgical lesion.

The analysis of our series of acute gall bladder infections at the Presbyterian Hospital between the years 1916 and 1930 shows among other data the following points:

SUMMARY OF FINDINGS IN 160 CASES OF ACUTE CHOLECYSTITIS OCCURRING IN 1860 CASES OF BILIARY TRACT DISEASE—9%.

Sex Incidence Age Incidence	Females 115 Females	Males 45 Males	Total
10-20	2	0	2
20-30	8	5	13
30-40	19	6	25
40-50	29	12	41
50-60	38	11	49
60-70	15	6	21
70-80	4	5	9
			160

Patients giving history of initial attack—25.

The others had had from 2 to 60 attacks of severe colic over periods of 1 month to 20 years.

The average temperature on admission—101 degrees.

Jaundice present in 22 cases.

CHOLECYSTECTOMIES—69

Died	7	Operative mortality—10%
Followed	47	Average follow-up period 40 months
Not followed	15	(Private cases)

Optimum result	444	31-66%
Symptomatic cure	84	39-83%
Symptomatic failure		5-10%
Hernia Ao		8-17%

CHOLECYSTOSTOMIES—91

Died	15	Operative mortality—16.5%
Followed	67	Average follow-up period 24 months
Not followed	9	(Private cases)
Optimum result	444	21-23%
Symptomatic cures	84	33-36%
Symptomatic failures ...		21-23%
Hernias		8-8.8%
Mucous fistulae		16-17.5%

Cases re-operated with cholecystectomy—20-22%

OPERATIONS

Cholecystectomy	69
Cholecystostomy	91
Choledochostomy	16

MORTALITY

With Cholecystectomy 7 deaths	10.0%
With Cholecystostomy 15 deaths	16.5%

COMPLICATIONS

Pneumonia	7
Peritonitis	7
Cholangitis	5
Diabetes	10
Thrombophlebitis	3
Uremia	6
Sepsis	4
Bacteriemia	2
Myocarditis	2
Acute Pancreatitis	2

MAJOR CONTRIBUTING CAUSES OF DEATH

Pneumonia	5
Vasomotor Collapse	4
Uremia	3
Gas Bacillus Infection	3
Sepsis	2
Pulmonary Embolism	1
Hemorrhage	1

PATHOLOGICAL FINDINGS

Stones in 130 cases	81%
Pus in Gall Bladder—118 cases	75%
Perforated Gall Bladder—30 cases	19%

Acute Pancreatitis—2 cases

BACTERIOLOGICAL FINDINGS

Cultures Taken	146 cases
No Growth	30 "
Positive Cultures	116 "
B. Coli	61
Enterococcus	19
Staphylococcus	14
Hemolytic Streptococcus	6
Typhoid	3
Paratyphoid	3
B. Proteus	4
B. Welchii	3
B. Oedematis	2
B. Friedländer	2
Pneumococcus IV	1
Pyocyaneus	1
B. Mucos. Cap.	1

Results in Cases with Disease Limited to the Gall Bladder

(As seen in a series of 400 consecutive biliary tract cases)

	Cholecystectomy			Cholecystostomy		
Acute	Cases	Compli-cated	Died	Cases	Compli-cated	Died
With stones	21	6	2	10	3	1
Without stones	2	1	0	3	1	1
	23	7	2	13	4	2
Chronic						
With stones	185	48	1	6	5	0
Without stones	56	20	0	8	2	0
	241	68	1	14	7	0

Total	Cases	Complicated	Died
Acute	36	11 (30%)	4 (11.1%)
Chronic	255	75 (29%)	1 (.039%)
	291	86 (30%)	5 (1.7%)

Results in Cases with Disease Not Limited to the Gall Bladder Requiring Common Duct Drainage

(As seen in a series of 400 consecutive biliary tract cases)
78 Cases—19 Deaths (24%)—50 Cases with Complications (64%)

	Cases	Deaths	Mortality Rate
Cholecystitis and Common Duct Stone the Only Lesions	35	5	14 per cent.

Acute	8	1	12.5 per cent.
Chronic	27	4	15 per cent.
Cholangitis Present	13	7	54 per cent.
Acute	4	4	100 per cent.
Chronic	9	3	33 per cent.
With Pancreatitis	28	5	18 per cent.
Acute Pancreatitis	3	1	33 per cent.
Pancreatic Lymphangitis	13	1	7 per cent.
Chronic Pancreatitis	12	3	25 per cent.

Only a surgeon operating on these acute biliary lesions has any conception of the difficulties encountered in these cases. The frequently associated obesity and septic state, the difficulties in anesthesia, the unsatisfactory and hurried operation because of the pathology found and the condition of the patient, the high mortality and the persisting symptoms and mucous fistulæ in so many gall bladder drainage operations necessitating secondary cholecystectomies —all these factors tend to convince the careful and conservative surgeon as well as the physician of the advantages of removing these foci of infection and potential trouble during the interval, at a time when the patient is in good physical condition and prepared for the operation of cholecystectomy.

ON THE NATURE OF FITS

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Delivered November 7, 1930, in the Friday Afternoon Lecture Series of The New York Academy of Medicine.

The diagnosis of epilepsy in most of its manifestations is easy; the comprehension of its nervous physiology is obscure and difficult. That epilepsy is not a disease is sure—it is a neural paroxysm in response to noxious stimulation—a broad description to clear the ground for better sight of the problem, that we may see the scope of our essay and the nature of our objective.

The idea of epilepsy given us by Hughlings Jackson is that it consists of an occasional sudden excessive violent discharge of some nerve center, sensory or motor; to which it would seem useful to add that the phenomena produced are independent of will and are accompanied by an alteration in the stream of consciousness. We shall see that even this ample cloak covers but sparsely all epileptic events which occur in such varied circumstances and in such a motley company of diseases that we are forced to the conclusion that the convulsive state and passing changes in consciousness cannot be any primary disease unit in themselves. They are but the expression of an innate or induced instability of nervous centers arising from many causes, operating probably through a common constant connecting mechanism. We must therefore seek out the nature of the agent which permits or elicits nervous explosion—a quest which must range from disorders of circulation to chronic poisoning and the protean appearances of anaphylactic shock. It is necessary to recognize, on the one hand, the essential unity of all episodes of impaired consciousness often accompanied by convulsion, and on the other, the amazing variety of conditions which may cause

such episodes. Such epileptogenous conditions are often fleeting: we must combat with vigor the portentous and often untrue prognosis so frequently saddled on such disorders by the public and many of our profession.

We are so familiar with epilepsy produced through renal disease and the poisonings of an uneasy pregnancy, that by granting such epilepsy special titles, we have obscured the fact that epileptic events in certain individuals follow hard on the heels of intoxications and are curable by the removal of the toxic material. Ignoring these facts, we arbitrarily classify cognate, often identical, phenomena in a category of disaster, whereby the diagnosis of epilepsy has become synonymous with a sentence to perdition. Epilepsy has been described by some of the most eminent writers on the subject as a condition characterized by recurrent convulsions and terminating in dementia. Surely this statement reveals but a fragment of one end of the spectrum of this disorder. True, the context usually attemps to adjust the ill balanced values set out by this description, but the context is apt to be difficult and the definition easy of quotation, the result being the unwisely restricted viewpoint so often taken of the condition and the often foolishly lugubrious outlook imposed on the subject and his friends.

In an attempt to comprehend the abnormal sequences comprised in a convulsive fit, we may first look at the neural mechanism involved and then regard the evidence on behalf of vascular disorders capable of permitting or producing the explosion, and lastly, speculate on the nature of the intoxicating agents initiating the train of events.

Epilepsy has been already described as an occasional paroxysmal discharge of a nerve center or group of centers occurring apart from volition and accompanied by interference with consciousness. Perhaps paroxysmal "impairment" might be a better word than "discharge," for many of the minor manifestations have such negative characteristics as to preclude the use of so positive a descriptive title. Even in the major seizure, the sequence of events

can be most readily understood as a sudden cutting out of the highest level, the cortex, allowing the lower neuronic levels to pour downward an ungoverned stream of tonic postural impulses—in fact, an abrupt rigidity of the decerebrate type. The subsequent convulsions which have secured, by their dramatic quality and their greater duration, more attention than I think they deserve, probably represent the gradual return of cortical control, incapable, at first, of ordered volitional action. I suggested at the beginning of this address that many diverse causative conditions must operate through a common mechanism to produce so constant a result, for my claim is nothing less than a qualitative unity and quantitative difference in the various manifestations of epilepsy, from the major seizure through petit mal, to migraine, to recurrent syncope and periodic endogenous emotional tempests. And in the gamut I should include, without equivocation, the voluminous mental states of unreality, and those prolonged and terrifying experiences known now as vasovagal attacks in which the patient is suddenly terror-stricken, suffers mediastinal oppression, has a feeling of tingling and swelling of the limbs, a sense of impending death and, losing precision of contact with his surroundings, experiences an acute transient seizure of cardiac hurry. Probably the mechanism of the major attacks will be revealed by a study of the manifestations of the minor attacks which will be found, I think, to be fragments of the fully developed seizure—just as we have fragments of the decerebrate posture produced by imperfect mesencephalic block, so we may have fragments of the great fit produced when abnormal conditions, probably of a vascular nature, are not sufficiently violent to unseat the entire cortical function. Such a "fragment" was demonstrated a few months ago in Bellevue Hospital by a young man, who for some years had been afflicted by sudden giving way of his legs on laughing. He had protected himself studiously against this cataplectic mishap by assuming a portentous gravity which seemed experimentally impregnable. However, one morning during rounds he was sitting on the edge of the bed with his

feet dangling and was unwarily provoked to a loud laugh by the remark of a neighbouring negro. Immediately following his emotional explosion, his hands were thrust out in a tonic spasm, the face was tonically convulsed, the lips were cyanosed, and he slid slowly off the bed on to the floor—during his descent the pupils were found fixed and enlarged. His small seizure ended with his fall, he picked himself up and stated he had had no loss of consciousness whatever. The time needed for the whole fit was, however, so short as to make it impossible to prove his statement nor could his reflexes be examined in the attack. Transient losses of the knee jerks, however, have been found by Kinnier Wilson and others in such cataplexies. This type of fit is probably a mesencephalic explosion; the irritated center being the affective control in the thalamus. The narcolepsies are in all likelihood, subthalamic expressions of the same order. A sudden overwhelming disposition to sleep descends on such patients, one of whom told how he would be overcome in a directors meeting in the middle of his own speeches and more oddly still in the middle of intensive courtship.

The vasovagal attack which I have described, if condensed in point of time, would be the same as the phenomena of many of the petit mal attacks, and I can personally vouch for its identity with the sensations felt when, having compressed my carotids and vagi, I lost control of my experiment and, a few seconds later, of consciousness.

Leonard Hill, more intelligently and successfully, compressed one carotid in himself: "the first effect was a sensation in the eye of the same side; then there followed a sensory march of formication down the opposite side of the body. This began in the fingers, spread up the arm and down the leg. Finally, clonic spasms of the hand occurred, accompanied by an intense feeling of vertigo and alarm." Whether clonic movements occurred in my own case, I do not know, as I was alone at the time and have since lacked inclination to repeat the experiment; the sensation in the hands and forearms for the few seconds

before loss of consciousness, was that of very powerful faradization; it was still present on my recovery but quickly passed away.

In connection with these considerations of vascular change, it is proper to remind ourselves of the cerebral phenomena produced by severe heartblock. Thomas Lewis finds that suspension of the mental functions is produced by a single period of asystole of from three to seven seconds' duration. In severer cases, where the pulse ceases for fifteen seconds or more, there occur cyanosis and stertor, and twitching of the face and limbs; fatal status epilepticus is by no means infrequent. In such cases there is a cessation of general circulation, but in the cats operated on by Stewart and Pike, cerebral and medullary circulation only were cut off. The immediate results were unconsciousness and bladder and rectal evacuation, severe tonic convulsions, then flaccidity (it must be remembered that midbrain circulation was obliterated); after the return of circulation there were clonic convulsions which prefaced resumption of consciousness and complete recovery.

In the ability of the emotions to cause severe vasomotor fluctuations and, in certain persons, syncope, and in certain others, headache or minor and major convulsions, we have an approach to a psychologic mechanism in epilepsy, which differs from that impressionistically sketched by Pierce Clark and others.

One cannot leave the subject of vasomotor instability in epilepsy without mentioning the remarkable case published by Osler of Raynaud's disease of the fingers, feet, ears, and nose, occurring during six winters and accompanied only during such seasons by severe epileptic convulsions.

Such events raise again the question of the existence of motor nerves in the cerebral blood-vessels. These were thought for long to be absent, but Gaskell's observations have demonstrated their presence; the muscular walls in

these vessels would seem evidence enough, but Biedl and Cushing have both made manifest that epinephrin blanches the brain as it does other tissues, and Wiggers has shown a definite diminution in the outflowing blood after the addition of epinephrin to the fluid circulating through the brain.

Forbes and Wolff working with Stanley Cobb have lately been able to demonstrate the contraction of pial vessels immediately after the stimulation of the cervical sympathetic nerve and their dilatation after the stimulation of the central end of the cut vagus.

Bernard Wortis in the Bellevue laboratory found blanching of mucous membranes and the cerebral cortex in cats during the moment preceding convulsions produced by the injection of camphor monobromide. MacDonald and Cobb using absinthe made the same observation. John Hartwell and myself were once able to observe the brain of a patient during a general epileptic fit: the exposure of the parietal cortex had been carried out under local anesthesia whereby consciousness and normal reflex activity were retained up to the moment of the attack. The initial sign of the seizure was a sudden whitening of the cortex which was no sooner remarked on than it was replaced by tremendous venous engorgement with protrusion of the brain beyond the level of the bone defect. This alarming engorgement, in which the previously blanched cortex became beet color and many of its veins swelled to half the diameter of one's small finger, was coincident with the tonic stage of the attack and the period of general clonic convulsion. The cortex failed to recover its normal appearance during the remaining twenty minutes of exposure, though its blood-red color was gradually mitigated in that period. The engorgement was apparently a part of the diffused cyanosis consequent on the general tonic spasm; the initial chalky appearance, however, must have been due to a vascular constriction which, I believe, was the proper beginning of the cerebral seizure.

Cushing has shown that faradic stimulation of the cor-

tex produces pallor of the gray matter and it might be well to bear his observation in mind when considering what exactly we mean by the spread of focal attack. Such spread is too slow to be explicable by the passage of a nerve impulse and might well be dependent on a quickly widening area of cerebral anemia. MacRobert and Feinier advanced an interesting theory to account for the fact that generalized epileptic seizures result more often from neoplasms in the temporosphenoidal lobes than from tumor formation in any other cerebral area. These observers believe that the topographical relation of the sylvian artery to these lobes renders the former susceptible to abrupt compression from the transient edemas surrounding the growth; such sudden diminished blood supply they believe, renders the cortical elements unstable, but as I have already suggested, the mechanism may be rather that the cortex is cut out by such an event and lower levels released by lack of adequate control.

Bealtie, Brow and Long, attribute the increased irritability of nerve centers, including the cerebral sympathetic centers, to asphyxia.

Indeed we may assert that the functional stability of nerve cells depends on adequate oxygenation. This may be lowered by reduction of blood-flow—possibly by central sympathetic stimulation—by vascular spasm or vascular sclerosis. Again, this relative cellular anoxemia may be produced by a film of fluid between the cell and its source of oxygen supply—the blood-vessel—this depicts the detail of the morbid process involved in the familiar epilepsies of alcoholism, uremic poisoning, and other forms of general or local brain edemas.

An instance of convulsions caused by cerebral edema, is a physician's child whose fits were coincident with urticarial attacks. These were found to depend on a sensitiveness to milk: both skin and cerebral expressions disappeared with the removal of the noxious protein.

Following up the notion that the initial tonic phase of

the grand attack is due to a stream of ungoverned impulses from the lower neuronic levels, we might remember that, in spinal convulsion due to strychnin poisoning, there is no initial tonic period and that clonic spasms occur from the beginning, whereas from midbrain intoxication by either tetanus or rabies, there occurs engravescent tonicity —lockjaw and risus sardonicus—or tonic convulsion as the first manifestation of disease. It would appear that the discharges of energy produced by strychnin poisoning would be included in the definition of epilepsy advanced by Hughlings Jackson, as would indeed those associated with rigor, simple shivering or that curious somatic tremor which some people experience after micturition. It is for this reason that I have thought it necessary for clinical purposes to make it clear that we consider as medically epileptic, only phenomena associated with alteration of consciousness. It is not desirable to say that, in what Gowers called the borderland of epilepsy, there is impairment of consciousness, for in the voluminous mental state there is often a feeling of expanded receptivity, a subjective enlargement of the orbit of awareness, a sense of prescience. Indeed, an extraordinary acuteness of reaction to sensory stimulation may occasionally be seen in epileptic persons.

Convulsions could be produced without fail in two patients under my observation. In one, a child, by flicking or sharply tapping the left side of the nose, in the other, an adult, by rubbing the dorsum of the left foot. The attack in the first case consisted in immediate dilatation of the pupils, tonic spasm of the arms and chest, respiratory arrest, cyanosis and slight frothing at the mouth, the whole lasting sometimes fifteen, sometimes twenty seconds, and ending in flaccid exhaustion after return to consciousness. There were no clonic movements in this child whom Dr. Jackson was good enough to come to the hospital to see and whose fit he regarded as a typical bulbopontine explosion.

In my second case, who was under the care of Sir

Farquhar Buzzard, the convulsions were general and followed the usual procedure of a major seizure. The right parietal cortex was exposed in this case without anything of note being revealed.

Jackson reported a case of a boy whose seizure was precipitated by touching his head, a sharp tap or sometimes combing his hair, would cause him to fall suddenly with paled cheeks but without loss of consciousness.

Some years ago, there was admitted to the neurological department of Bellevue Hospital, a boy aged 17 years whose history stated that for twelve months he had been subject to instant falling to the ground on hearing a sudden sound —a horse pawing the ground in a quiet street, a boy whistling behind him—would be enough to produce cramp in the left leg and a quick fall in which he was always bruised. He denied any loss of consciousness and certainly to onlookers, gave for a long period, no evidence of such. However, after two years of these events, frequently repeated, he had a major convulsion which came on after the slamming of a door and was preceded, as were the other attacks, by a feeling of cramp in the leg. These larger seizures did not entirely replace the fallings after sudden sounds. I induced both types of attack with ease, but did so rather seldom as he would fall violently on his face and, so innocent was he of any reflex of defence, I feared for his safety.

This is an example of the reflex acusticomotor epilepsy described by Oppenheim; for many months, the boy was thought by some competent men to be hysterical. His first fall occurred at school when he was trying to solve a tough problem at the blackboard; his crayon broke and he fell sharply to the ground. His infantile history, however, supplied an organic background if not an organic explanation. Three weeks after birth there was still present a very large caput succedaneum over the right occipitoparietal area. This was opened and a blood clot evacuated. There was said to have been a bone defect in this area at that time. Symptoms of meningitis are said to have been present during the first two weeks of life. When five

months old he had a general convulsion; later, he began to have jerky movements when falling asleep and on waking; in his mother's words: "The left leg would come up and the left shoulder and body go forward; the movement would occur eight or ten times but varied in number and intensity." These hypnogogic events lasted with lessening severity for fourteen years; two or three years later they were replaced by the recurrent episodes just described.

The parietal cortex was exposed. A great thickening of the dura was discovered, produced by an old organized, subdural clot. The attacks have become rare since the operation, and hypersensitiveness to sounds uncommon. This case has been told at some length because of its intrinsic oddness, but also because it shows how cerebral impulses can be short circuited by trauma and react quickly to stimulation by discharge to vasomotor changes.

Trauma, certainly, can render a brain area unstable. Severe skull and brain injuries in War were found to have given rise to subsequent convulsions in 4 per cent of 25,000 cases under review. Wortis has shown in our laboratory that the injured cortex explodes with a smaller dose of a standardized convulsant (camphor) than does the intact cortex. Wilder Penfield (*Brain* Vol. 53 part 2 p. 118) has pointed out the effect of scar tissue in cortical wounds, and believes that cicatricial traction on surrounding blood-vessels is inevitable, and is the cause, by vasomotor reflexes, of the convulsion—the type of which will depend on the location of the scar.

It is needless to discuss here the exact relationship between migraine and epilepsy; they are both found in persons of neuropathic type and heritage; both diseases often occur in different persons of the same stock and very many instances are reported where they existed at different times in the same patient. In a case of my own, the left-sided headache was ushered in by a sensation of flashing lights in the right temporal field, numbness in the right hand, and ingravescent hemianopia, alexia, and aphasia, all of which became practically complete. The subsequent

headache lasted for some hours during which the patient behaved rationally. Following the seizure he had complete amnesia for the period of the attack with the exception of its first half-hour.

Migraine has been reported as alternating in three patients with family periodic paralysis—but it would be futile to list here the numberless forms of equivalents which have been described and are entirely familiar to this audience. Earlier in this address I used the term the spectrum of epilepsy. My general purpose has been to suggest that in this spectrum are many colors which merge insensibly into each other; uremia and eclampsia of pregnancy, the fits of general paralysis and brain tumor are there; the spasmophilic conditions of infancy are also to be thus grouped, including certain forms of tetany associated with alteration of consciousness; the chorea of pregnancy may be in this spectrum also, and—not very deep in the ultraviolet sector—are probably some cases of major hysteria, the motor phenomena of which suggest the temporary abandonment of a cortical for an infracortical mechanism.

In fine, as we have come to allocate destructive lesions of the brain by their focal signs so we may associate the many types of epileptic expression with appropriate neural areas, which have been the objective of vascular attack; for example petit mal is almost certainly an alteration of consciousness from frontal disorder, complex visual, auditory and psychical hallucinations from disorder in the temporosphenoidal lobes, gross color fits or twinkles from disorder in the occipital poles, myoclonic epilepsies from striatal implication, the cataplexies and narcolepsies from thalamic and subthalamic disorder, and the great fit as has been pointed out is a sudden decerebration with a cutting out of the cortical totality followed by a reviving paroxysm of the motor areas.

The researches of Bronson Crothers and Howard Smith (*Amer. Jorun. Obst, and Gyn.* pp. 19; 374, 1930) into the frequency of central nervous system injury occurring in the act of birth illuminates an organic morbidity which

has received insufficient attention. You will remember that in 100 spontaneous deliveries there was bloody fluid in 11 per cent; in 50 low forceps deliveries 24 per cent of the cistern fluids were bloody. In five cases of breech extraction there was blood in 40 per cent of the fluids, and when in 5 cases the first stage of labour had lasted over 24 hours it was found that 60 per cent of the fluids were contaminated with blood. The fluids of Cesarean babies were all blood free.

Olken (*Journ. Nerv. and Ment. Dis.* Nov. 1930, p. 544) has been able to show distortion of the cutaneous capillaries in babies delivered by forceps and in those of normal birth; whereas no such distortion could be made out in 17 Cesarean section babies. It was also found that microscopic capillary hemorrhages were found throughout the pia arachnoid after experimental convulsions in dogs.

These clinically indistinguishable birth traumata of the brain may well act directly as agents for the reduction of adequate cell oxygenation in the brain or indirectly by an interference with cerebral vasomotor control through the central sympathetic. At all events such birth injuries must often be capable of lowering the threshold of neural explosion to a point accessible to stimulation by metabolic products, toxic maybe, but innocuous in a subject with a higher threshold of nerve cell irritability.

The act of birth assumes then a highly hazardous and romantic aspect. Death has been called the greatest adventure of life but there is no adventure in a certainty; the real gamble for all of us lies in our entrances, our future lies behind us, and with this knowledge we may be able to do good preventive medicine even in the field of epilepsy with assistance from our obstetrical colleagues.

Once the minute injury has been done our efforts in treatment must reasonably be directed, first to raising the threshold of neural explosion and second, to getting rid of all detonating stimuli likely to reach it.

The first is achieved by the continued use of small doses

of neural sedatives, bromide of course, but still better, phenobarbital. Pavlow (*Conditioned Reflexes* 1927: 10) has taught us how inevitably the neural reflexes become conditioned and to allow fits to occur because of fear of "drugs" is obsessional superstition.

The next is to rid the oversensitive organism of noxious stimulation. To this end have been directed most of the modern therapeutic systems. Lane and Reed when permitted enthusiastically resected the entire colon; other men more conservatively have been content with a bacteriological revision of the great intestine, by providing proper diet, peristalsis and flora. Geyelin in 1921 studied the effects of periodic starvation on epileptics and found that severe fasting considerably reduced the number of attacks and in a small number eventually caused their arrest. From his work has been evolved the plan of producing by a diet very rich in fats, a ketosis similar to that produced by starvation: a child of 58 lbs. on this system should begin by 60 grms. of carbohydrate, 129 grms. of fat and 25 grms. of protein in the 24 hours.

No one program, however, will be successful or even moderately successful in all cases. Nor could we expect one design to be of service against so varied a congery of symptoms.

An approach to real knowledge may lie in the chemical study of sympathetic nervous system stimulants and toxic protein split products produced by proteolytic ferments.

The humoral pathology—to use Zabriskie's phrase—of the disease must be made comprehensible by anaphylactic studies, and when these things have all been added unto us we must remember to treat the patient as a whole, to adjust his environment and his problems, to arouse his interests, keep fired his zest for living, inculcate discipline and wise habits, and as far as possible allow him to live normally within himself—with fair weather in his soul—and as happily as maybe also in the Society of his choice.

EXTRACT FROM THE MINUTES OF THE COUNCIL OF JANUARY 28, 1931 IN REGARD TO DR. HIGHMAN

The Committee on Professional Standards received complaints against Dr. Walter J. Highman because of his name appearing in an advertisement of Woodbury's soap. The matter was thoroughly investigated by the Committee before which Dr. Highman appeared. The report of the Committee was presented to the Council, which after very thorough discussion and consideration adopted the following resolution in accordance with their authority as given in Article IX, Section 1 of the Constitution:

RESOLVED that Dr. Walter J. Highman be reprimanded and suspended for a period of six months and that a notice of this action be published in the Academy Bulletin.

STANDARDS FOR OXYGEN THERAPY EQUIPMENT

COMMITTEE ON PUBLIC HEALTH RELATIONS

Until recently, the employment of oxygen tents and similar equipment for oxygen or carbon dioxide therapy in the private practice of medicine outside of hospitals has been restricted almost wholly to a few physicians who are especially expert in the application of this form of therapy as a result of experience gained in hospital practice. Its application to the treatment of pneumonia and other conditions characterized by an interference with the proper oxygenation of the blood has become so extensive that commercial agencies have now found it profitable to rent equipment to physicians.

The Committee on Public Health Relations has been urged to take steps to acquaint physicians of the community with the information necessary to guide them in the employment of this form of therapy. It is not enough that the indications for the treatment be understood. A physician who expects to use the equipment must understand the mechanical features of the tent employed and know how to judge its mechanical efficiency.

In employing an oxygen tent or other apparatus for the treatment of pneumonia, it should be borne in mind that, by improving the oxygenation of the blood and reducing cyanosis, the rate and character of the respirations are improved, the patient is made more comfortable, and is saved a large amount of work. If the oxygen content of the tent is decidedly above or below the optimum, or if the atmosphere is too hot or too moist, the patient may be made very uncomfortable and the therapy is then capable of doing more harm than good.

I. With regard to the *air conditioning,* it is of utmost importance that:

(1) The tents should have a capacity of at least eight cubic feet.

(2) The temperature inside of the tents should be maintainable at or below that of the outer air.

(3) The concentration of oxygen should be 45 per cent (±5) when oxygen is fed at not more than 8 litres per minute during maintenance. Continuous use of concentrations of oxygen above 70 per cent are harmful. For this reason it is necessary that the concentration of oxygen be measured at frequent intervals during the time it is used by the patient.

(4) The carbon dioxide content should be not more than 1 per cent unless specifically desired.

(5) The relative humidity should be below 50 per cent, regardless of inside or outside relative humidity or temperature, taking into consideration the fact that the patient in the tent may add as much as 1000 c.c. of water to the atmosphere each day.

(6) The tent should be serviced in such a manner as to provide at least fifteen air changes per hour.

(7) All tents should be provided with thermometers and suitable hygrometers readily readable from the outside.

(8) Tents should be equipped with an apparatus to test the oxygen and the carbon dioxide concentration.

II. With regard to *sterilization of the tents,* all tents should be sterilized after each use by scrubbing inside as well as outside with soap and water. This work could be facilitated and made more effective if the tents were made of double-faced material. After scrubbing, the tent should be dipped in a solution of 1 to 10,000 of bichloride of mercury for five minutes. To prevent incrustation, the tent

should be washed down with water after immersion in the bichloride of mercury. In hospital practice, an alternate method would be the dipping of the tent in a 70 per cent solution of ethyl alcohol. An inexpensive method of sterilization is to expose the tent for 45 minutes to formaldehyde vapor followed by 15 minutes' exposure to ammonia vapor.

III. With regard to the *fire hazard*:

(1) All tents should be conspicuously stamped, "NO FLAMES, NO SPARKS, DANGER."

(2) Oxygen gauges should be tested for accuracy before release and be conspicuously labeled "DANGER, DO NOT OIL."

(3) For the window of the tent, only cellulose acetate or other non-inflammable material may be employed, and cellulose nitrate or celluloid should be specifically forbidden.

IV. With regard to *testing*:

(1) The basic loss of oxygen of each tent should be determined before rental or sale and stated on an attached tag.

(2) Oxygen should not be misbranded so as to suggest that it is other than industrial oxygen.

V. A less efficient method of oxygen therapy, which is much less expensive, is the use of the nasal catheter or the nasal tube inhaler with a calibrated gauge to fit on a high pressure tank. These should be sterilized before rental or sale, and in the case of rental, sent in a sealed container.

DEMONSTRATION OF OXYGEN THERAPY

The exhibit of oxygen therapy held in the exhibition room of the Academy Building on February 9th and 10th proved as successful as had been anticipated. Twenty-one exhibitors demonstrated oxygen tents or other types of equipment employed in oxygen therapy. In addition, two types of portable oxygen chambers were shown as well as respirators and other apparatus used for resuscitation. It is estimated that about 1200 physicians visited the exhibition during the two days. Each afternoon and evening, talks were given by authorities on the use of oxygen.

The exhibit was planned to stress the need for adequate methods of oxygen administration, and to acquaint physicians as well as inventors and manufacturers of oxygen tents, and the commercial firms which engage in the rental of tents and oxygen equipment, with the necessity of appreciating the conditions to be attained or avoided, and with the real menace of faultily designed or operated equipment. Minimum requirements were listed and emphasized, and it was pointed out that a patient in urgent need of oxygen may be made more restless and uncomfortable, and be actually harmed, by being placed in a tent in which oxygen concentration, temperature and humidity are not measured and controlled.

The administrators of hospitals in the metropolitan area as well as individual physicians had the opportunity to compare many types of oxygen tents, reducing valves, and other pieces of apparatus, and to acquaint themselves with their relative advantages and disadvantages for hospital and private use. It was brought out that the use of oxygen has been greatly limited by excessive cost. As a remedy for this situation a reduction of charges was urged, based on standardization and simplification of equipment, in anticipation of wider demand. At the same time the

use of the less expensive nasal inhaler was advocated for the purpose of partially relieving oxygen need, and for the milder cases or where the expense of a tent cannot be met, as well as when tents are not tolerated and an oxygen room is not accessible. The large variety of tents within one room enabled manufacturers to compare the relative advantages and disadvantages of their apparatus with that of others, so that improvement, simplification and reduction in cost will undoubtedly result shortly because of this opportunity. Stereomotorgraphs which showed successively and continuously, at a controlled rate, slides describing the principles of oxygen therapy, illustrated the value of this method of instruction.

The necessity for adequate sterilization of the tents and for protection against fire hazards was emphasized. The representatives of the City Department of Health who visited the exhibit were impressed with the necessity of some official action leading to new regulations of the Department in order to guard against cross infection through improper cleansing and sterilizing.

As a result of the exhibit four commercial firms have entered the business of renting tents to physicians. These firms, as well as others who sell oxygen, will also rent reducing valves so that oxygen may be purchased in high pressure tanks at a fraction of its cost when supplied in the less efficient low pressure tanks on sale at the corner drug stores. Firms which sell oxygen also agreed to discontinue the practice of misbranding their tanks as "medical oxygen" and selling them at a price greater than that charged for the same oxygen when purchased for commercial purposes. They also agreed to maintain proper ethical practices in the rental of equipment so that the same rental charge will be made whether the bill is paid by the patient directly or by the physician.

The success of the exhibit called attention to the possibility of using the exhibition room of the Academy for similar demonstrations designed to acquaint physicians with new types of therapeutic equipment and with the technique of newer forms of therapy.

THE FIFTH ANNUAL

ART EXHIBIT

EXHIBITION OF WORKS

IN THE PLASTIC AND GRAPHIC ARTS

BY AMERICAN PHYSICIANS UNDER THE AUSPICES OF THE NEW YORK PHYSICIANS ART CLUB

WAS HELD AT

THE NEW YORK ACADEMY OF MEDICINE

FEBRUARY 14 TO MARCH 14, 1931

LIBRARY NOTES

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Paris, Masson, 1930, 488 p.

REQUEST FOR MINUTES

The Committee on Library would be very pleased to receive as gifts or, failing that, on deposit for safe keeping, the past minutes of any medical society or medical club. Time and again we have found that such minutes have been of great help in looking up the history of a particular man or branch of medicine. We all recognize that with the change of secretaries minute books are often lost. We have volumes of such material here already, some received as donations and others merely deposited as loans.

PROCEEDINGS OF ACADEMY MEETINGS
FEBRUARY

Stated Meetings

Thursday Evening, February 5, at 8:30 o'clock
Program presented in coöperation with the
Section of Obstetrics and Gynecology

ORDER

I. Executive Session
Election of Fellows
II. Paper of the Evening
Endocrine diseases: their diagnosis and therapy, Robert T. Frank
Discussion opened by, Charles C. Lieb, Charles R. Stockard

Thursday Evening, February 19, at 8:30 o'clock

The Fifth Harvey Lecture
"Possible Immunological Reactions With Plants"
E. M. East
Harvard University, Cambridge
Alfred E. Cohn, President Harvey Society
Dayton J. Edwards, Secretary Harvey Society

Section of Dermatology and Syphilology

Tuesday Evening, February 3, at 8:30 o'clock

ORDER

I. Reading of the Minutes
II. Presentation of Cases
a. Cases from Vanderbilt Clinic
b. Miscellaneous cases
III. General Discussion
IV. Executive Session

Section of Surgery

Friday Evening, February 6, at 8:30 o'clock

ORDER

I. Reading of the Minutes
II. Presentation of Cases
a. Resection for volvulus of a sigmoid megacolon, Carnes Weeks
b. Resection of right colon for idiopathic hypertrophic colitis, Edward J. Donovan
III. Symposium on Hemorrhoids
a. Medical aspect, John L. Kantor
b. Surgical treatment, Jerome M. Lynch
c. Injection treatment, Frank C. Yeomans
d. High frequency current treatment, William Bierman (by invitation)

IV. General Discussion

Section of Neurology and Psychiatry
Tuesday Evening, February 10, at 8:30 o'clock

ORDER

I. Reading of the Minutes
II. Clinical Presentations
From the neuro-endocrine service of the Neurological Institute and Vanderbilt Clinic
a. The Laurence-Brindle-Moon Syndrome, John McD. McKinney
b. Hyperglycaemia in hypopituitarism, with glandular treatment, Josephine H. Kenyon (by invitation)
c. Pluriglandular compensatory syndrome, Walter Timme
III. Papers of the Evening
a. Disorders induced by injuries to the pituitary and hypothalamus, Philip E. Smith, Ph.D., Columbia University (by invitation)
b. The hormonal cause of premenstrual tension, Robert T. Frank
Discussion, Charles R. Stockard, Walter Timme
IV. General Discussion
V. Executive Session

Section of Pediatrics
Wednesday Evening, February 11, at 8:30 o'clock

ORDER

I. Papers of the Evening
Dental Nutrition
a. Dental physiology, Charles F. Bodecker
b. The influence of vitamins upon dental nutrition, Percy Howe, (by invitation)
c. Calcium and phosphorus as factors in development, Henry C. Sherman (by invitation)
Discussion, Alfred Walker (by invitation), Oscar M. Schloss

Section of Otology
Friday Evening, February 13, at 8:30 o'clock

ORDER

I. Reading of the Minutes
II. Presentation of Cases
III. Report of Cases from the Ear Clinics of Drs. Arthur B. Duel, John B. Rae, John R. Page and E. Prince Fowler of the Manhattan Eye, Ear & Throat Hospital.
Introductory remarks, Arthur B. Duel
a. Simple mastoidectomy complicating cervical Pott's Disease, Arthur F. Warren (by invitation)
b. Secondary mastoidectomy complicated by an apparent parotid abscess, J. S. Manning, Jr. (by invitation)
c. End results in 20 cases of primary skin grafts in radical mastoidectomies, Daniel S. Cunning (by invitation)

d. Sinus thrombosis complicating chronic mastoiditis, Wright MacMillan (by invitation)

e. 1. Secondary simple mastoidectomy with choked discs and severe cerebral symptoms

2. Temporosphenoidal lobe abscess complicating chronic otitis media purulenta, Henry M. Scheer

f. Acute mastoiditis followed by cerebellar abscess, operation and recovery, H. Clifton Luke

g. 1. Hernia following sinus and jugular operation

2. Double mastoidectomy complicated by retropharyngeal abscess, bilateral sinus thrombosis and necrosis of labyrinth on one side, Charles W. Byrd

h. Acute labyrinthitis, Lester Mead Hubby

i. 1. Post-operative radium treatment in carcinoma of mastoid and middle ear

2. Primary radium treatment in carcinoma of external ear, G. Allen Robinson

j. Report of a case of cerebellar abscess which followed an operation for mastoidectomy, sinus thrombosis and jugular resection on opposite side, John R. Page

k. 1. Malignant angiomatous polyp with sequestrum of mastoid tip (Avellus-Jackson Syndrome)

2. Deformity of ear and face from lupus erythematosis, treated by tuberculin, E. Prince Fowler (by invitation)

Case discussed by Howard Fox, Charles W. Williams

Discussion, Arthur B. Duel, John B. Rae, John R. Page, E. Prince Fowler (by invitation)

SECTION OF OPHTHALMOLOGY

Monday Evening, February 16, at 8:30 o'clock

ORDER

I. READING OF THE MINUTES

II. PRESENTATION OF CASES

a. A case of tubercular keratitis-fistula of the cornea; repair, Ben Witt Key

b. Angioma venenosum of the orbit, Joseph Stanton Hory

c. A case of traumatic pulsating exophthalmos (post-operative), Martin Cohen

d. A cyst of the iris, A. E. Town (by invitation)

III. DEMONSTRATION

A new method for studying the normal and pathological surface anatomy of the eye, Mark J. Schoenberg

IV. PAPERS OF THE EVENING

a. Proptosis as a diagnostic problem (lantern slides), Ralph I. Lloyd

b. The circulation of the aqueous, Jonas S. Friedenwald, Baltimore (by invitation)

V. GENERAL DISCUSSION

VI. EXECUTIVE SESSION

Section of Medicine

Tuesday Evening, February 17, at 8:30 o'clock

A purely clinical program has been arranged for this meeting, at which cases of unusual clinical interest will be presented and discussed.

ORDER

I. Presentation of Cases
   a. Splenomegalic polycythemia, complicated by amebiasis, Louis F. Bishop, Jr. (by invitation)
   b. Polycythemia vera, three cases, Joseph E. Connery
   c. Polycythemia vera due to chronic carbon monoxide poisoning, George Baehr
   d. The therapeutic use of oxygen over a period of six months in a patient with pulmonary fibrosis in cardiac insufficiency, Alvan L. Barach
   e. A case for diagnosis, Joseph Hajek
   f. Enterogenous cyanosis, Franklin Hanger (by invitation)
   g. Heart block in a young man without symptoms, Edwin T. Hauser
   h. A case of general lymphadenopathy for diagnosis, Harry A. Solomon

II. Discussion
   Knut H. Houck (by invitation), W. W. Palmer, Dickinson W. Richards, Jr. (by invitation), Emanuel Libman, H. S. Patterson, Nellis B. Foster, Harlow Brooks, Lewis A. Conner, John Wyckoff

Section of Genito-Urinary Surgery

Wednesday Evening, February 18, at 8:30 o'clock

ORDER

I. Reading of the Minutes

II. Paper of the Evening
   Certain congenital malformations of the genito-urinary tract: their diagnosis and treatment, Hugh H. Young, Johns Hopkins Medical School (by invitation)
   Discussion, Benjamin S. Barringer, Henry G. Bugbee, Oswald S. Lowsley, A. T. Osgood, Nathaniel Rathbun, J. Sturdivant Read, A. R. Stevens, J. J. Valentine

III. General Discussion

IV. Executive Session

Section of Orthopedic Surgery

Friday Evening, February 20, at 8:30 o'clock

ORDER

I. Reading of the Minutes

II. Papers of the Evening
   a. The treatment of acute gonorrheal arthritis by the injection of air, William B. Porter (by invitation)
   b. The treatment of severe injuries to the ligaments of the knee joint, H. Page Mauck (by invitation)

III. General Discussion

SECTION OF OBSTETRICS AND GYNECOLOGY

Tuesday Evening, February 24, at 8:30 o'clock

ORDER

I. READING OF THE MINUTES

II. PRESENTATION OF CASES

a. Ovarian pregnancy—case report, Anthony Wollner (by invitation), Alfred Hellmann

b. Stricture of the female urethra—case reports, Salvatore di Palma, H. D. Furniss, L. M. Kahn

c. Report of a case of uretero-vaginal and vesico-vaginal fistula successfully repaired in 1920, H. Dawson Furniss

III. PAPER OF THE EVENING

The elective classical caesarian section as a preventative of obstetric morbidity, Edward A. Schumann, Philadelphia (by invitation)

Discussion, George L. Brodhead, George W. Kosmak, Hervey C. Williamson, H. B. Mathews, Edward C. Lyon, Jr., Frederick Holden, Prof. Graff of Vienna

IV. GENERAL DISCUSSION

V. EXECUTIVE SESSION

SECTION OF LARYNGOLOGY AND RHINOLOGY

In Conjunction with

THE NEW YORK GASTROENTEROLOGICAL ASSOCIATION

Wednesday Evening, February 25, at 8:15 o'clock

ORDER

I. PAPERS OF THE EVENING

SYMPOSIUM ON "THE ESOPHAGUS"

a. Diverticula, Frank H. Lahey, Boston (by invitation)

b. Strictures, Gabriel Tucker, Philadelphia (by invitation)

c. Cardiospasms, Charles J. Imperatori

II. DISCUSSION

John E. Mackenty, Armistead C. Crump, Mervin C. Myerson

NEW YORK ROENTGEN SOCIETY

In Affiliation with

THE NEW YORK ACADEMY OF MEDICINE

The regular February meeting was not held on February 16 for the reason that the Society held a two day conference in Baltimore on February 6 and 7, arranged by the Baltimore Roentgen Society.

ROSS GOLDEN, President

J. BENNETT EDWARDS, Secretary

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

Under the auspices of

THE NEW YORK ACADEMY OF MEDICINE

Wednesday, February 18, at 8:15 o'clock

I. On the Cortical Hormone of the Adrenal Gland, W. W. Swingle and J. J. Pfiffner

II. A Precipitant for Material in Liver Active in Pernicious Anemia, R. West, M. Howe, and H. D. Dakin

III. Specificity of Reactions Produced by Injection of Urine from Pregnant Cows upon Immature Female Guinea Pigs, G. N. Papanicolaou

IV. Study of the Virus of the Common Cold and its Cultivation in Tissue Medium, A. R. Dochez, K. C. Mills and Y. Kneeland, Jr.

V. Relationship of Sera and Spinal Fluids to Agglutination and Flocculation by Dyes, L. Rosenthal and O. S. Hornick

VI. Demonstration of a Tumor Inhibiting Substance in Filtrate of Rous Chicken Sarcoma, and in Normal Chicken Sera, M. J. Sittenfield, B. A. Johnson and J. W. Jobling

VII. The Gastric Hunger Mechanism. II. The Effect of Diet, M. G. Mulinos

VIII. Further Note on the Enumeration of Blood Platelets and Red Blood Cells, A. E. Casey (introduced by L. Pearce)

IX. Delayed Differential Counting of the White Blood Cells by a Modified Supravital Technique, A. E. Casey and P. D. Rosahn (introduced by L. Pearce)

PEYTON ROUS, *President* A. J. GOLDFORB, *Secretary*

NEW YORK PATHOLOGICAL SOCIETY
In Affiliation with
THE NEW YORK ACADEMY OF MEDICINE
Thursday Evening, February 26, at 8:30 o'clock

ORDER

I. PAPERS OF THE EVENING

a. Congenital esophageal stenosis due to aberrant ductus arteriosus, Henry Rascoff, Mendel Jacobi

b. Spongioneuroblastoma; a tumor formation associated with tuberous sclerosis, J. H. Globus (by invitation), H. Selinsky

c. The occurrence of malignancy in radio-active persons, Harrison S. Martland

d. Serum and plasma bilirubin: a comparative study of 100 cases, Rubin Finkelstein, Mendel Jacobi

II. READING OF THE MINUTES

FELLOWS ELECTED MARCH 5, 1931

Alan R. Anderson ..... 237 East 20th Street
Renfrew Bradner ..... 6 East 85th Street
George A. Cashman ..... 65 East 66th Street
Augustus Harris ..... 306 Park Place, Brooklyn
Maurice Lenz ..... 1049 Park Avenue
J. Arnot MacGregor ..... 121 East 60th Street
Beryl Holmes Paige ..... 622 West 168th Street
Frank H. Peters ..... 400 East 58th Street
Herman Selinsky ..... 345 West 86th Street
Barbara B. Stimson ..... 25 Claremont Avenue
Marjorie Lord Strauss ..... 108 East 91st Street

DEATHS OF FELLOWS OF THE ACADEMY

CHARLES BENNET BRODER, M.D., 240 East 15 Street, New York City; graduated in medicine from Cornell University Medical College, New York City, in 1905; elected a Fellow of the Academy February 5, 1914; died, February 15, 1931. Dr. Broder was a Fellow of the American Medical Association, a member of the County and State Medical Societies and Laryngologist and Otologist to People's Hospital.

DEATH OF MR. JOHN S. BROWNNE

RESOLUTIONS PASSED AT THE STATED MEETING OF THE ACADEMY HELD MARCH 5, 1931

Mr. John S. Brownne consulting librarian of the Academy, died at his home in New Jersey on Friday, February 27, 1931 at the age of 76.

Mr. Brownne became librarian and general superintendent in 1880 and served the Academy in that capacity continually until 1925. Throughout this period of 46 years the history of the Academy was intimately identified with the life experience of Mr. Brownne.

"Under the zealous efforts of Mr. Brownne, the Library increased rapidly in volume and in importance. Soon after the date of his appointment, Dr. John S. Billings was made Librarian of the Library of the Surgeon General's office in Washington, and thus at once Mr. Brownne

found himself confronted by a powerful rival, as shown in the competition between them for desirable accessions. In these competitions Mr. Brownne often outbid his rival funds being provided by enthusiastic members or by their generous friends.

"The growth of its Library, according to the record, eloquently proves the esteem in which it was held. The total books in 1875 amounted to 21,720; in 1880 to 25,000; in 1900 to 49,830, and in 1910 to 80,790. In 1926 there were 139,320 books, 98,685 pamphlets, while 1,500 were journals subscribed for, making a total of 239,505 titles.

"If the fame accorded to Dr. Billings for the upbuilding of the second greatest medical library in the world is justly deserved, then equal credit is due to our own Librarian for the success of his life work in creating the Library of the Academy as it stands today, the third greatest of its kind."

RESOLVED that the Academy hereby records its deep sense of loss in the death of Mr. John S. Brownne, consulting librarian of this Institution to which he devoted a lifetime of service, and be it futher

RESOLVED that a copy of this minute and resolution be published in the Bulletin of the Academy and sent to the members of his family.

1931
STANDING COMMITTEES OF THE ACADEMY

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Rolfe Floyd
Richard T. Atkins
Ward J. MacNeal
DeWitt Stetten
Harbeck Halsted
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Alfred T. Osgood
Harry M. Imboden
George M. MacKee

Emanuel D. Friedman

Committee on Library
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Alfred F. Hess
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James Alex. Miller, *Chairman*

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and
The Chairman of each Section

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